R.M.LOESER

A TEXT-BOOK OF LEGAL MEDICINE AND TOXICOLOGY

EDITED BY

FREDERICK PETERSON, M.D.

President of the New York State Commission in Lunacy; Clinical Professor of Psychiatry, Columbia University, New York; General Consultant to the Craig Colony for Epileptics, Sonyea, New York

AND

WALTER S. HAINES, M.D.

Professor of Chemistry, Pharmacy, and Toxicology in Rush Medical College, Chicago; Professorial Lecturer on Toxicology in the University of Chicago

In Two Volumes

Containing about 1500 Pages, Fully Illustrated

VOLUME II

PHILADELPHIA, NEW YORK, AND LONDON

W. B. SAUNDERS & COMPANY

1904

ELECTROTYPED BY
WESTCOTT & THOMSON, PHILADA

PRESS OF
W. B. SAUNDERS & CO., PHILADA

CONTRIBUTORS TO VOLUME II.

WILLIAM T. BELFIELD, M. D., CHICAGO.
Associate Professor of Surgery in Rush Medical College, in Affiliation with the University of Chicago; Professor of Genito-Urinary and Venereal Diseases in Chicago Policlinic.

CHARLES GILBERT CHADDOCK, M. D., ST. LOUIS.
Professor of Diseases of the Nervous System, Marion-Sims-Beaumont College of Medicine, Medical Department of St. Louis University.

JOSEPH F. DARLING, A. M., LL.B., NEW YORK CITY.

EDWARD P. DAVIS, A. M., M. D., PHILADELPHIA.
Professor of Obstetrics in the Jefferson Medical College, Philadelphia; Visiting Obstetrician to the Jefferson and Philadelphia Hospitals.

CHARLES A. DOREMUS, A. M., M. D., PH. D., NEW YORK CITY.
Formerly Professor of Chemistry and Toxicology, Medical Department, University of Buffalo; Member of the Medico-Legal Society; Associate Member of the Massachusetts Medico-Legal Society.

W. A. NEWMAN DORLAND, A. M., M. D., PHILADELPHIA.
Associate in Gynecology, Philadelphia Polyclinic; Assistant Obstetrician to the Hospital of the University of Pennsylvania, etc.

J. T. ESKRIDGE, M. D.,[1] DENVER.
Late Consulting Alienist and Neurologist, Arapahoe County Hospital; late Neurologist to St. Luke's Hospital, Denver, Col.

MARSHALL D. EWELL, A. M., M. D., LL. D. (Univ. of Mich.), CHICAGO.
Professor of Elementary Common Law and Medical Jurisprudence in the Chicago-Kent College of Law; formerly Lecturer on Medical Jurisprudence in the University of Michigan.

LEONARD FREEMAN, B. S., A. M., M. D. DENVER.
Professor of Surgery, Denver and Gross Medical College; Surgeon to Denver City and County Hospital, St. Joseph's Hospital, St. Luke's Hospital, and Jewish National Hospital, Denver, Colo.

A. L. GOLDWATER, M. D., NEW YORK CITY.
Lecturer on Chemistry and Toxicology to the New York Board of Pharmacy Institute; Instructor in Laryngology, New York School of Clinical Medicine.

[1] Deceased.

WALTER S. HAINES, M. D., CHICAGO.

Professor of Chemistry, Pharmacy and Toxicology in Rush Medical College, in Affiliation with the University of Chicago; Professorial Lecturer on Toxicology in the University of Chicago.

CHARLES HARRINGTON, M. D., BOSTON.

Assistant Professor of Hygiene in the Harvard Medical School; Associate Member of the Massachusetts Medico-Legal Society.

JAMES W. HOLLAND, M. D., PHILADELPHIA.

Professor of Medical Chemistry and Toxicology, Jefferson Medical College, Philadelphia; Member of the American Philosophical Society, Fellow of the College of Physicians, Philadelphia.

REID HUNT, PH. D., M. D., BALTIMORE.

Associate Professor of Pharmacology and Toxicology, Johns Hopkins University, Baltimore.

WALTER JONES, PH. D., BALTIMORE.

Associate Professor of Physiological Chemistry and Toxicology, Johns Hopkins University, Baltimore.

CARLOS F. MACDONALD, A. M., M. D., NEW YORK CITY.

Professor of Mental Diseases and Medical Jurisprudence in the University and Bellevue Hospital College, New York: Consulting Physician to Manhattan State Hospitals; Consulting Physician to the Psychopathic Hospital and Laboratory of the New York Infirmary for Women and Children; Ex-Superintendent of the Auburn and Binghamton State Hospitals for the Insane; Member of the American Medico-Psychological Association, etc.

HAROLD N. MOYER, M. D., CHICAGO.

Assistant Professor of Medicine in Rush Medical College, in Affiliation with the University of Chicago.

OSCAR OLDBERG, PHARM. D., CHICAGO.

Dean of the School of Pharmacy of Northwestern University, Chicago.

ALBERT B. PRESCOTT, M. D., PH. D., LL. D., ANN ARBOR.

Director of the Chemical Laboratory, Professor of Organic Chemistry, and Dean of the School of Pharmacy in the University of Michigan.

JEROME PROBST, PH. G., LL. B., of the Chicago Bar, CHICAGO.

JEROME H. SALISBURY, M. D., CHICAGO.

Professor of Internal Medicine, Chicago Clinical School; Assistant Professor of Chemistry in Rush Medical College, in Affiliation with the University of Chicago.

GEORGE KNOWLES SWINBURNE, A. B., M. D., NEW YORK CITY.

Surgeon to Good Samaritan Dispensary, New York City.

VICTOR C. VAUGHAN, M. D., PH. D., SC. D., LL. D., ANN ARBOR.

Dean and Professor of Hygiene and Physiological Chemistry in the Department of Medicine and Surgery of the University of Michigan.

EDWARD S. WOOD, A. M., M. D., BOSTON.

Professor of Chemistry in the Harvard Medical School; Member of the Massachusetts Medical Society; Member of the Massachusetts Medico-Legal Society, etc.

PREFACE.

THE object of the present work is to give to the medical and legal professions a fairly comprehensive survey of forensic medicine and toxicology in moderate compass. We believe this has not been done in any very recent work in English. A number of manuals of limited size and scope have been presented on the one hand, and on the other certain systems of legal medicine of almost encyclopedic dimensions. Both find fields of great usefulness; but there is still left a broad ground intermediate between the two which we trust the present work will fill, and it was in this hope that the book has been planned and executed.

With few and wholly unimportant exceptions the articles composing the two volumes have been inserted without change by the editors. This has been done in order that the responsibility for statements of facts and opinions may be authoritatively placed upon the individual contributors—a matter of much moment in legal proceedings. In doing this we are aware that we have occasionally sacrificed unity of plan and harmony of views, but the advantages, especially to the legal profession, of individual responsibility we believe much more than compensate for these defects.

As the ordinary English weights and measures and the Fahrenheit thermometer scale are still the only ones easily understood by the majority of courts and juries, we have generally used these measures, wholly or in conjunction with their equivalents in metric weights and measures and the Centigrade scale. This rule, however, has not been followed in the description of purely chemical tests and processes, as the metric system is practically universally employed in connection with them.

In the names of drugs and chemicals we have followed the nomenclature and, with slight modifications, the spelling of the United States Pharmacopeia, believing that by adhering to so authoritative and well known a standard as this much confusion will be avoided and a not infrequent source of perplexity to lawyers and physicians eliminated.

The work is divided, for convenience of reference, into two sections, Part I. and Part II., the latter being devoted to Toxicology, and all other portions of Legal Medicine in which laboratory investigation is an essential feature.

Our thanks are due to the many distinguished men who have aided us in the production of the work by their valuable contributions; and we are greatly indebted to the publishers, Messrs. W. B. Saunders & Co., for the unfailing interest they have shown in the book, and for the numerous courtesies they have extended to us in its preparation.

FREDERICK PETERSON,
WALTER S. HAINES.

CONTENTS OF VOLUME II.

PART I.

PART II.

CONTENTS OF VOLUME I.

PART I.

(CONTINUED.)

MALINGERING AND THE FEIGNED DISORDERS.

From the beginning of the human race affections of a surgical nature have been simulated—by cowards, to avoid the dangers of combat; by beggars, to excite compassion; by rogues, to attain their ends; and by others, for purposes as varied as they are sometimes obscure. Even children are not exempt. The little boy who suddenly acquires a lame knee and cannot attend school, and the little girl who sprains her wrist in time to avoid an hour's practice on the piano, are as certainly malingerers as is the soldier who feigns contractured fingers in order to escape military service. Animals are often skilled in deception. The woodcock, in order to draw attention from her young, will simulate a broken wing; and the painful limp of an unwilling horse will miraculously disappear when his head is turned toward home.

Feigned surgical affections, with the exception of such as are due to hysteria, are comparatively so rare in private practice, and real ones so common, that we are apt to be caught off our guard and do not recognize the fraudulent symptoms when they appear. Hence the necessity for the surgeon to acquire an idea not only of diseases which may be feigned, but of the motives and characters of those who are most likely to feign them. He should be familiar with every form of surgical trouble in all its variations in order to avoid mistaking a real disease for one which is simulated, which is far more culpable than overlooking a simulation. It is better that many spurious affections be regarded as genuine than that one genuine case should be declared spurious. If we could pass in review the legion of unfortunate wretches whose real sufferings have given rise to neglect and maltreatment under the impression that they were feigned, we should employ more time and care before coming to conclusions in similar cases in the future. It is also well to remember that many instances of simulation occur in those who, from some cause or other, are not in good physical condition.

Opportunities for fraud in the domain of surgery are far fewer than in that of medicine. It is easier to simulate a colic, for instance, than a hernia. Nevertheless, deceptions are commonly enough met with, particularly in the army and navy and in hospitals and prisons; while pension examiners and physicians to accident-insurance companies are constantly harrassed by the most obscure forms of fraud.

In former times feigning of surgical diseases was much more prevalent than now. For this there were various reasons. Beggars were more numerous and importunate; in fact, begging assumed almost the dignity of an industry, and deception was practised as an art. Apparent

deformities, nauseous sores, and frightful tumors, draped by the rags of filthy mendicants, were seen in profusion on the streets, and there were even those who made a business of manufacturing cripples.

Most of these things, however, were mere stage tricks which required only close observation for their detection, and were calculated to deceive the passer-by rather than the surgeon.

In the army and navy there were formerly many more malingerers than there are to-day, malingering becoming so common at times that the most stringent laws were enacted for its suppression. This was particularly true when conscription was in vogue. Individuals resorted to all sorts of deception and self-mutilation in order to escape entering the service or to obtain a discharge. So-called "epidemics" of incontinence of urine appeared among young men. Teeth were filed off and extracted by the wholesale in order that men should become unfitted to bite off the ends of the old-fashioned cartridges. Many resorted to the cutting or shooting off of fingers and the severing of tendons. Simulations of hernias, hydroceles, contractures of joints, ulcers, and swellings of various kinds were often seen.

A reason for the lessened prevalence of such frauds is the marked improvement in the service. Men enter voluntarily instead of being impressed, and they are treated more like human beings and less like slaves. The food and hygienic surroundings are better, and personal liberty is greater. In fact, where much malingering is found, there is generally a cause, which should in justice be removed if possible.

Another reason is that malingering has been rendered easier of detection by such instruments of precision as the clinical thermometer, the microscope, the laryngoscope, the ophthalmoscope, etc. Surgical anesthesia is also of immense value in the diagnosis of obscure cases.

It is often better to reserve judgment for a time until opportunity can be gained to study the case in all its bearings. It may be useful to appear to believe all that is said until the patient, secure in the apparent success of the fraud, finally commits himself. If he can be convinced of the hopelessness of gaining his ends, he may sometimes be inclined to give up the deception. In order to accomplish this most effectively it is better that the examination be conducted privately, for the larger the audience, the more stubborn the subject. Sometimes the mere threat of severe treatment, such as amputation or the cautery, will effect a cure. Violent measures should, as a rule, be avoided, as they often do little good and it is always possible that the trouble may be real. A soldier has been known to walk for eighteen months with his back bent double and his fingers within two inches of the ground, in spite of the most severe treatment, rather than give in; and, on the other hand, the writer has seen a stoker on an ocean-steamer, who refused to work on account of real weakness and pain, killed by a ducking, followed by exposure, under the impression that he was a malingerer.

The simulator is apt to give an incorrect history of his disease, or couple with it some ridiculous symptom, especially if such things be cleverly suggested by the examiner. Care is requisite, however, in

this connection. I once saw an old, deformed leper on the island of Molokai who earnestly claimed that on wet days he felt the bacilli of leprosy enter the tips of his right fingers, crawl slowly up the arm, around his neck, and down the left arm. In spite of his foolish statement there could be no question as to the reality of the disease.

It is often best not to treat actively a simulated surgical disease—ignore it as far as possible. Especially is this true of hysteria. It is a fact, however, that those who are feigning are often averse to treatment, especially if severe, while the possessor of a real and troublesome affection grasps eagerly at anything that offers relief.

It is difficult to classify simulated surgical lesions. Although many attempts have been made, none of them seems to be entirely satisfactory. According to the writer's view, hysteric diseases should stand in a group apart, although they are seldom so placed. Hysteria is really a form of disease; and although its manifestations are not genuine diseases, they are, strictly speaking, not simulated. Individuals are hysteric not because they wish to be, but because they cannot help it. A working classification, differing somewhat from that which is generally accepted, is as follows:

A. SIMULATED AFFECTIONS (abnormal conditions which are not real).
   1. *Feigned Affections.*—Those which are entirely fictitious, whether they are known lesions or inventions of the imagination.
   2. *Factitious Affections.*—Those which have been produced by the patient, but which are claimed to be due to disease.
   3. *Exaggerated Affections.*—Whether the exaggeration be wholly assumed, or whether the existing condition be really exaggerated by voluntary means.

B. SELF-PRODUCED AFFECTIONS (INJURIES, ETC.).—These are what they appear to be, the only fraud being in the assertion of the individual as to their causes.

C. HYSTERIC AFFECTIONS.

SIMULATED AFFECTIONS.

1. Feigned Affections.—Pain and Tenderness.—These are purely subjective symptoms, for the existence of which the surgeon must, for the most part, accept the assertions of the patient. Hence simulation is easy and the fraud difficult to detect. Old deformities, scars, injuries, etc., offer an excellent basis for fictitious pains, and enable their possessors to live in comfort in our charity-hospitals. And yet we are compelled to believe these impostors until we can prove them to be frauds. The most flagrant examples of this form of feigning are met with among applicants for pensions and for insurance, and among those injured in accidents and assaults, where damages are desired.

The general appearance of the patient is of importance. Those who have much pain will soon show the characteristic effects of suffer-

ing and loss of sleep. If the administration of a small dose of morphin, without the patient's knowledge, readily produces sleep, much pain cannot be present. Pressure on the sore spot during natural sleep may easily expose a fraud. It is often possible so to divert the patient's attention by examining other portions of his body, by plying judicious questions, etc., that the alleged tender and painful part may be quite roughly handled. A system of espionage is as valuable here as it is in the detection of other feigned diseases. The relation of the cause to the alleged effect should be taken into consideration, a mere scratch on the body of a malingerer often giving rise to the most severe apparent suffering.

Concussion of the Brain.—This is often feigned by those who wish to exaggerate the effects of a blow or pretend that they have received one. It is a favorite trick of boxers in order to escape further punishment. In real concussion there are a pale, moist skin, feeble pulse, superficial respiration, and perhaps nausea, which are not present when concussion is feigned.

Wry-neck.—This is not commonly simulated, and can easily be detected. When fraud is attempted, the sternomastoid muscle of the side opposite to which the head is turned is tense, especially when efforts are made by the surgeon to correct the deformity. The eyes can be moved toward the side from which the head is rotated with difficulty only, while in the real disease this has been rendered easy by habit. The close observer will also notice in true torticollis an asymmetry of feature which it is impossible to simulate. Like other fraudulent contractures, wry-neck is apt to be forgotten when the patient is drunk or excited, and the muscles, of course, become relaxed during sleep or when under an anesthetic.

Lateral Curvature of the Spine.—Although this subject will be considered under Railway Spine, a few words will be in place here. In true scoliosis the vertebræ are rotated on their longitudinal axes, so as to bulge out the ribs on the convex side and wing the scapula. A secondary curve is likewise present, which serves to bring the body into the line of gravity. In feigned scoliosis, which occurs almost invariably in the dorsolumbar region, the spine is not twisted on its axis, but merely bent to one side, and there is no compensatory curve, no bulging of the ribs, etc., although there is a marked wrinkling of the skin which has not had time to adjust itself to the new position.

Non-hysteric Contractures of Joints.—Contractures are so easily imitated, and are apparently so disabling, that they are often seen among those who wish to deceive. Injury, rheumatism, neuralgia, or some obscure nervous affection may be assigned as a cause. Atrophy of adjacent muscles and swelling of the joint may be produced by prolonged tight bandaging, while local irritating applications may counterfeit inflammatory changes. The suspicions of the surgeon should be aroused when the joint exhibits no pathologic changes ; or when the nature or extent of the injury or disease appears to be inadequate to have produced the apparent effect. A scar, for instance, intentionally

or accidentally produced, may be found on closer inspection to be but skin deep. The presence of a nervous trouble may often be excluded by the absence of characteristic symptoms. Contractured muscles, which frequently exist, may have no relation whatever to the seat of a supposed or actual neuralgic pain.

The fact must not be lost sight of, however, that trivial injuries, such as a slight wrench or bruise, at times precede serious tubercular joint-disease, which may manifest itself at first by muscular contractures alone. This is particularly true of deeper seated joints, such as the hip. When coxitis has existed for some time, its discrimination from feigned disease should be easy. Patients may simulate contractures to a certain extent, but not the absolute fixation of the joint, the muscular atrophy, the dropping and obliteration of the gluteal fold, the worn and peevish expression, etc. Those bent on deception sometimes betray themselves by the incongruity of their symptoms —by complaining of pain, for instance, when the knee is struck, but not when the blow is delivered against the heel. Pain in the knee, so common in real disease, is often neglected in the simulated form.

In the more superficial joints the presence of definite spots of tenderness is strongly indicative of tubercular foci, in contradistinction to a general sensitiveness of the part.

When contracture is said to be due to causes which have produced real shortening of the muscles, binding of ligaments, or union between the joint-surfaces, the fraud is easy of detection, as movement is suddenly checked during manipulation without evidence of voluntary muscular action, while an anesthetic quickly clears up any obscurity in diagnosis. Hence these forms of ankylosis can hardly be successfully feigned, and it is practically only the acute muscular variety which calls for serious investigation. General anesthesia is here of little use, as it merely demonstrates that the contractures are purely muscular, without indicating either the presence or the absence of real disease.

Simulated contractures are apt to increase when under direct observation, and the muscles are always tense, while in real joint-deformities they may be lax. Investigations made while the subject is asleep, drunk, or excited, or when imagining himself alone, are often of value. Suddenly pushing the patient off his balance, so that the affected limb must be straightened in order to prevent a fall, has assisted the writer in demonstrating fraud. Arousing men from their slumbers with an alarm of fire, supported by a smell of smoke, has been known to effect astonishing cures. Some of the older army surgeons ordered the suspected individual to balance himself on his sound leg on a high pedestal. In order to prevent falling as the muscles began to tire, the contracted leg would be put down alongside its fellow.

A weight suspended by a cord will quickly straighten simulated contractures, but it must not be overlooked that recent contractures from disease may also be overcome in this manner, although less promptly.

An Esmarch strap wound around the limb above the affected joint,

so as to compress nerves and muscles tightly, will often serve to relax the part and expose an impostor.

The application of the thermocautery, while not out of place in many forms of real joint-disease, often rapidly effects a cure in fraudulent affections.

Hernia, Hydrocele, Varicocele.—There was a time when these diseases were frequently feigned by the injection of air or fluids beneath the scrotal or inguinal tissues, but such deceptions are seldom, if ever, met with at present. The feel of the part, the emphysematous crackling, and the absence of the well-known and characteristic signs of the trouble in question should establish a diagnosis at once, provided that the examination is at all thorough. Some men possess the power of drawing their testicles into the external abdominal rings, so as to present the appearance of hernia to a superficial observer. This may frequently be seen in children.

That which is most often feigned in hernia is pain, and the fraud is difficult to detect, unless the symptoms are grossly exaggerated. It should be borne in mind, however, that the pain and discomfort of a hernia are more commonly located nearer the umbilicus, at the attachment of the mesentery, than at the seat of the rupture.

Fistula in Ano and Fistula in Perineo.—These forms of fistula have been simulated, though not often, by cauterizing artificially produced wounds. The appearance of the part, together with the careful use of a probe, should settle such a question at once. In the case of the urethra, the absence of a stricture and of leakage of urine will be conclusive.

Hemorrhoids and Prolapsus Ani.—That these diseases should have been successfully feigned by inserting intestines and inflated bladders of animals into the anus seems almost incredible, and yet such instances have been recorded. Careful inspection must detect the deception at once.

Incontinence of Urine.—The simulation of incontinence was at one time so popular among soldiers that so-called "epidemics" resulted. In the genuine disease urine constantly dribbles from the urethra, instead of being emitted at intervals in a stream with more or less force. There is also no effort of expulsion, while in the feigned form, if the patient be stripped, the surgeon will observe contraction of the abdominal muscles. If the individual, while voiding urine, be suddenly commanded to stop and does so, it is certain that he possesses at least fair control of his bladder.

Most authors state that an infallible test for incontinence consists in the passage of a catheter at some odd moment when the patient is not suspecting such a manœuver,—on waking from a long sleep, for instance,—and if more or less urine be found, the trouble is fraudulent. Such advice is misleading. When there is an enlarged prostate, there is often a quantity of residual urine, and also, at times, incontinence.

Another test formerly employed by army surgeons consisted in tying a cord about the penis. In case of real trouble the penis soon swelled

from accumulated urine. Fouderé once had occasion to tie up a large number of soldiers in this ridiculous manner.

The fact that in real incontinence the glans and meatus are pale and sodden in appearance is usually dwelt upon. Although this is generally true, it is not a reliable diagnostic point. Neither is it of much importance that the clothing may smell of decomposed urine. Positive indications against malingering would be the presence of a stricture, an enlarged prostate, ammoniacal urine with bacteria and pus, or symptoms of some nervous affection with vesical paralysis.

Urinary Calculi and Gravel.—Hysteric patients, and occasionally others, sometimes pretend to have passed stones and gravel, which they exhibit in proof of their statements. If the suspicions of the surgeon have been aroused, he can easily detect the fraud by a chemical examination of the stones, which are usually but ordinary pebbles.

Hematuria.—Blood has often been mixed with the urine in order to feign this symptom. But if the patient is requested to urinate in the presence of the surgeon, or if a microscopic examination is made, there can be little difficulty in exposing the deception. The blood of a fowl may be recognized by the oval shape of its corpuscles. The Indian fig is said to color the urine blood-red, while the use of cantharides produces actual hematuria.

Ecchymoses and Contusions.—These are often feigned by staining the skin, but close inspection will usually reveal the fraud without difficulty. Sometimes the stain may be washed or rubbed away. The well-known changes in color of a contusion should be looked for after sufficient time has elapsed. A true bruise is deepest in color at its center.

2. Factitious Affections.—**Ulcers.**—The feigning of ulcers is a very ancient and very common trick, practised by the hysteric as well as by beggars, malingerers, and others. All sorts of mechanical and chemical irritants have been employed in their production. A favorite method has always been to bind a copper coin tightly against the shin. The metal was erroneously supposed to be the active agent, while in reality it is the pressure. After erosion of the surface has once been obtained it is easy to keep up the ulceration by mechanical irritation. Such ulcers, of course, refuse to yield to ordinary treatment. When they are falsely said to be of long standing, the cheat may be detected by noting the absence of callous margins, of edema, and of ecchymosis.

It may be necessary to incase a limb bearing an ulcer in plaster-of-Paris in such a manner that the patient cannot obtain access to the part. Ordinary bandaging, even if the bandages are sealed, does not always answer. Individuals have been known to keep up the irritation by inserting pins through the dressings. At one time factitious ulcers became so prevalent in the English army that it became necessary to lock up the affected limbs in wooden boots in order to circumvent the malingerers.

Beggars may sometimes succeed in deceiving the public by fastening

pieces of liver, etc., to their shins, but physicians could never be misled in this manner.

Tumors.—It is not easy to simulate a tumor, although it has been done. It must be a stupid surgeon, or a careless one, who is deceived into regarding as a cancer a piece of cow's liver glued to a breast, or a sponge saturated with red fluid and inserted into the bowel. Such frauds have been practised successfully, however, by beggars, and for the purpose of giving color to miraculous cures.

It is otherwise, however, with certain muscular contractures, which may closely resemble tumors. Some people have such control over the recti muscles that they can contract each segment at will, leaving the others unaltered. A few years ago a colored man traveled about the country, from one medical college to another, who possessed this power. By throwing the muscular swelling against the aorta, an apparent pulsation was produced, the exhibitor asserting that he had "thrown his heart into his abdomen." So-called "phantom tumors" of the abdomen are far less common than formerly, for many of them, such as intermittent hydronephrosis, have been found to be real. A few, however, still occur, most of which have a hysteric basis. They are variously due to local intestinal paresis with unequal gaseous distention, to muscular contractures of the abdominal walls, etc. In their detection the recognition of the hysteric state, combined with local symptoms, is usually sufficient. Gaseous tumors are tympanitic, and muscular contraction can be felt to be external to the abdominal cavity.

The situation of such a tumor as regards some muscle, together with the fact that the contraction cannot be long maintained, especially during sleep and anesthesia, should prevent a mistake in diagnosis.

Patients may claim that swellings, due to injuries or to more or less acute inflammatory processes, are tumors. When an untruthful history is cleverly given, the diagnosis may be rendered obscure. The main point lies in suspecting, from the circumstances or from the character of the patient, that something is wrong, when a little time, coupled with intelligent observation, will serve to clear up a doubtful case.

Diffuse swellings and localized tumors of the breast, due to hysteria, are described by Tourette. Marked hyperesthesia combined with other hysteric manifestations renders a diagnosis possible, although confusion is sometimes produced by edema and consequent ulceration. Pressure upon the tumor may cause a hysteric attack. Operations have been undertaken with the idea that a malignant tumor was present.

Dislocations.—Dislocations are very difficult to feign. Rarely, however, individuals are met with who possess the power of dislocating their joints at will; and there are others whose bones are so loosely articulated, perhaps through previous injury, that but slight force is required to throw them out of place. Such persons may utilize their anomalous construction in exhibiting before medical students, in gaining admittance to hospitals or in obtaining money from insurance companies or mutual-benefit associations.

If the dislocation is claimed to be recent, the absence of swelling,

ecchymosis, etc., would expose the fraud. When the affection is said to be of long standing, diagnosis is more difficult. In those who dislocate their joints at will, however, the luxations are always intracapsular, and hence not so complete as those which have been produced by violence and in which the capsule has been ruptured. Observation of the patient for a length of time, particularly when he is not aware that he is being watched, will expose the fraud, as it cannot be kept up indefinitely. It is often impossible to reduce old dislocations, while the form under discussion can be easily replaced. The joints which are most commonly dislocated at will are the shoulder and hip.

Fractures.—Patients have been known to move a broken bone continually in order to retard or prevent union, and they sometimes remove the splints and twist the parts out of shape. This is usually done in order to obtain grounds for a malpractice suit. Many reputable physicians who have done everything in a case that it was possible to do have been seriously annoyed in this manner.

A favorite trick of "hospital birds" is to complain of pain and tenderness at the site of some old fracture. In the absence of redness or swelling suspicion may be entertained of the genuineness of the trouble, especially if the individual overdoes his part, which is apt to be the case. The writer has seen an old ununited fracture of both bones of the forearm successfully passed off as recent, the member being carefully put up in splints by a hospital physician.

Gonorrhea.—An apparent gonorrhea may be produced by applying caustics to the prepuce, or by injecting irritating solutions within the urethra. The absence of the gonococcus upon microscopic examination, although not conclusive, is certainly suggestive. Sealing up the penis in such a manner as to prevent the application of irritants will rapidly effect a cure.

Swelled, discolored, and inflamed limbs may be caused by ligatures, perhaps very small ones, which should be looked for if the case is suspicious. It is said that acute swelling of the face and head, and even elephantiasis of the leg, have been simulated by this means.

That **varicose veins** can be produced by ligatures is perhaps possible, but the fraud is certainly uncommon, although varicose veins which already exist may thus be exaggerated in size.

It should be remembered that **atrophy of a limb** can be brought about by tight bandaging; and that various deformities have been caused in this or similar ways, particularly in ancient times. The feet of Chinese women and the flat skulls of certain Indians bear testimony to this fact.

3. Exaggerated Affections.—It is with exaggerated diseases that the surgeon is most often brought in contact. A slight sprain is magnified into a serious injury by the holder of a policy in an accident-insurance company; an old and painless hernia becomes an unbearable burden to the owner who desires admission into a hospital; a wound, long since almost forgotten, is nursed into violent activity by the applicant for a pension. There is no end to the exaggeration of symptoms

in connection with broken bones, deformed joints, injuries, tumors, ulcers, inflammations, etc. The physician must be continually on his guard, in high life as well as in low, among ladies and gentlemen as well as among beggars and rogues.

Exaggeration can usually be detected by observing the rules and principles elsewhere given in this article, although there are many cases which are manifestly never discovered. We are all prone to exaggerate our ailments at times, and we are seldom caught in the act.

SELF-PRODUCED INJURIES.

Self-inflicted wounds are not feigned diseases, although they are often considered under this heading. The wound itself is real enough, and is exactly what it appears to be, the only uncertainty being as to how it was made and who made it. It is a mere question of truth and untruth, in which there is no simulation of something which does not exist, as there is in the case of a wound which is produced by the patient and which he pretends is a chronic ulcer. Self-mutilations most frequently occur in connection with the military or naval service, particularly the former. At one time it was not uncommon for men to disable themselves in various ways in order to avoid being impressed into the army or navy. In Egypt, for instance, it was said to have been difficult to find a thoroughly able-bodied young man. Self-mutilations are now met with particularly in battle, where a comparatively slight wound made by the soldier himself may send him to the hospital and save him a more severe one made by the enemy.

Individuals who have attempted suicide and failed are at times tempted, through shame, to assert that the wounds were made by others. Through motives of revenge, in order to make others regret some real or fancied ill-treatment, self-injuries may be inflicted. Even children have frequently been known to practise this deception.

Various other instances may arise where it is of supposed advantage to simulate injuries. For instance, a gentleman who desired to be favorably considered financially by a relative pretended to have been wounded in an attempt to shield the relative from an assault. A colored cadet at West Point, who had failed in his studies, attempted to enlist sympathy by inflicting superficial injuries upon himself and binding himself to his bed, as though he had been roughly handled by his fellow-students. We not uncommonly read of bank-officials or of men in whose care valuables have been intrusted being found wounded and gagged, and the valuables gone. Sometimes it has been shown that these men have purposely injured themselves. It is not impossible, with a little practice, for a man to bind himself apparently quite securely. People who are hysteric, especially women, often injure themselves, with the idea, perhaps, of gaining notoriety and sympathy.

In detecting these various impositions it is of importance to discover a motive of sufficient weight, and to consider whether it was likely for the injury to have been produced in the manner or degree pretended.

It would scarcely be thought possible, for instance, that a man could stab or shoot himself in the center of the back; or that he would produce a fracture of the skull, or sever an important artery, unless he were attempting suicide.

Self-inflicted wounds, except suicidal ones, are nearly always superficial and comparatively trivial, while the apparent symptoms which accompany them are usually out of all proportion to the cause. It is very frequently the left side of the body which is injured, because most people are right-handed (Dr. J. N. Hall, of Denver, has called particular attention to this point). One who cuts his throat begins with vigor on the left side, but pain and faintness often cause him to lessen the pressure on the knife before the right side is reached. A man who is assaulted usually grasps the assassin's knife and receives one or more cuts across the hands in consequence, while the hands of the man who injures himself are intact.

Fraud may at times be detected by noticing the discrepancy between the injury and the assigned cause. A soldier who says his fingers were blown off by a gun, and whose stumps are, nevertheless, evidently severed by some sharp instrument, is open to the gravest suspicion.

In gunshot injuries the presence of powder-stains, burns, or scorched clothing would prove the falsehood of the statement that the injury was inflicted by some one at a distance, although individuals have been known to wrap a cloth about the part before shooting themselves. It should be noticed whether the bullet fits the patient's own weapon or not. If the pretender be requested to place himself in the position in which he was when injured, he is often unable to do so. It is a curious and confusing fact, that should be remembered, however, that when the barrel of a firearm, at least one of small caliber, is held very close to the body, the bullet may not penetrate the skin at all, or at least the damage may be greatly lessened. It is also true in such a case that powder-grains are not apt to enter the skin, only a superficial blackening and scorching of the cuticle resulting.

Women have been known to bruise and lacerate themselves, generally to a moderate extent, in order to give color to their stories of attempted assaults upon their virtue.

Some persons, generally hysteric women, exhibit a curious tendency to mutilate themselves by inserting various substances beneath the skin. Hundreds of needles have been removed from beneath the cuticle of such individuals in the course of a few weeks or months. The number of needles, together with the fact that most of them are found on the left side of the body, should excite suspicion. The account of an interesting case went the rounds of the medical journals a few years ago. A woman, apparently in good health, was continually troubled with the exfoliation and extrusion of pieces of bone from the left forearm. Hundreds of pieces of bone were obtained and preserved. A careful examination of the specimens later on, however, showed them to be chicken-bones.

HYSTERIA.

Any of the diseases and symptoms which have been mentioned, as well as many others, may be simulated by the hysteric, and the means of diagnosis are often the same. Ulcers or lateral curvature of the spine, for instance, are just as apt to be feigned by those who are hysteric as by those who are malingering. The general symptoms of hysteria, however, can usually be depended upon to help out in the diagnosis; although we should by no means consider that, because a woman has a lump in her throat and is subject to "spells," all the manifestations of disease which she presents are necessarily hysteric.

The subject of **hysteric joints,** however, is of sufficient importance to merit special consideration. The resemblance to real disease is at times so great that the most noted surgeons have gone so far as to perform an arthrotomy upon a perfectly normal joint. The general symptoms of hysteria are usually present, although they may be so obscure as readily to be overlooked. Patients with hysteric joints are often so sweet, good, and long-suffering that this alone may arouse the suspicions of the watchful physician. Hyperesthesia of the affected region is very characteristic. The sufferer complains of pain as soon as the skin is lightly touched, and even before; but if the attention is strongly attracted in some other direction, the joint may often be quite firmly pressed without exciting discomfort. There is, in the great majority of instances, no physical evidence of disease on inspection, palpation, or measurement; but occasionally a truly perplexing case is encountered in which a certain amount of heat, redness, and swelling exists. The reflexes remain equal on opposite sides of the body, although they may be exaggerated. A genuine hysteric hydrops has rarely been described. The diagnosis must be based upon the presence of other evidences of hysteria, together with local hyperesthesia and the exclusion of all forms of real disease. The absence of fever and other constitutional disturbances which accompany actual inflammation is always suggestive.

Hysteric contractures are sometimes met with, and may be confusing in respect to diagnosis. Although any joint may be affected, the most common situation is the tarsus, the foot assuming the position of equinovarus. Here, again, the hysteric temperament is in evidence, and the causes are inadequate to the effect. The writer has seen a woman, for instance, who slightly sprained her ankle during a quarrel with her husband, shortly after the injury suddenly acquire a strongly marked equinovarus, which was maintained for many weeks. Her disposition was resigned and sweet throughout, and the sympathy of friends was strongly elicited. Ten days' isolation completed a rapid cure.

Another case which was seen in a medical clinic in Berlin was one in which there was such marked flexion of the knee-joint that the

patient, a little girl, was compelled to walk with a strong limp upon the tip of her toe. This condition had existed for several years. Stationing himself on the opposite side of the room, the clinician took his watch in his hand, fixed his eyes upon the patient, and told her that in five minutes she would place her heel on the floor and walk without a limp, which she did.

The treatment of these cases consists, first of all, in the treatment of the general hysteric condition—in appropriate foods and tonics, in change of surroundings and conditions, and in change of mental atmosphere, if this be possible. Isolation from sympathizing friends is of the utmost importance, and should be tried in every obstinate case. Little, if any, attention should be given the affected joint itself—all forms of corrective apparatus, local applications, etc., only seem to make matters worse by keeping the attention concentrated upon the affected part. Sometimes, however, a well-planned bogus operation is of great benefit, as happened in the experience of a surgeon of the writer's acquaintance, who removed an imaginary frog from a woman's stomach by means of a fictitious gastrotomy, performed through a skin-deep laparotomy. A prominent surgeon of New York cured a hysteric tumor of the brain by making an incision in the scalp only, although he was subsequently sued for obtaining money under false pretenses. To what extent such means are justifiable in attempts to cure is a question which every one must decide for himself, according to the circumstances of the situation. The electric brush and the thermocautery are resorted to in hospitals and institutions of various kinds, and at least a temporary cure is often effected by their use. In private practice they will usually not be tolerated, and considering that the results are mostly but temporary, it is questionable if we are in general justified in using such painful means. Hypnotic suggestion may be of the greatest value.

FEIGNED MENTAL AND BODILY DISORDERS.

Feigned disease is not a product of modern civilization. Sacred and profane histories contain references to cases of feigning, both of mental and bodily diseases. The instruments of precision and the more intimate knowledge of diseased processes of the present day enable us to detect without much difficulty simulation by the ignorant and unskilled. Fortunately for the medical expert, feigning disease is largely limited to this class. But it occasionally happens that persons of experience and great intelligence, and even physicians skilled in the knowledge of disease, adopt dishonest methods to escape condign punishment or to obtain undue advantage.

Feigning Mental Disorders.—Insanity is most frequently feigned by those who have committed some criminal act; occasionally by others who have entered into some legal contract, the results of which they wish to avoid on the theory of irresponsibility; less often by en-

terprising newspaper-reporters in order that they may be sent to an insane asylum and have an opportunity to write up the management; and, least frequently of all, by the hysteric for the purpose of gaining sympathy and notoriety.

In all cases of feigned insanity there is a motive for simulation. This should never be lost sight of by the physician in investigating every case of alleged mental disorder, and when no motive for feigning is revealed by a careful inquiry into the history and acts of the individual, no suspicion of feigning is justifiable. On the other hand, the acts of many insane, before they are apprehended and sent to an asylum, are constantly bringing them into collision with the law. A motive for feigning then, in a case of alleged insanity, is no proof of simulation. It should be borne in mind that some insane persons feign a different form of insanity from that from which they are really suffering, so that the detection of simulation of a mental disorder by a person alleged to be insane does not justify the conclusion that he is sane. Under these circumstances such a conclusion should be arrived at only after a most searching investigation has failed to detect any form of mental aberration. While it is true, as many have asserted, that in criminal cases insanity is rarely assumed until after the commission of the crime and the apprehension of the criminal, yet we must remember in epileptic, alcoholic, and traumatic amnesia that the individual cannot claim innocence until he is accused of acts of which he has no knowledge. Such a person's insanity, if it is due to epilepsy or alcoholic indulgence, may have existed previously, but attention has not been attracted by it if no overt act has been committed before. Taylor states that a person does not feign to avoid suspicion. This is true in the vast majority of cases, but if a criminal felt sure that circumstances were such that he ultimately would be suspected of a crime of which he was guilty, he might feign insanity before he was accused. Feigned insanity is almost always sudden in its onset, while mania, melancholia, paranoia, and dementia, the forms of insanity most frequently simulated, develop gradually. Second attacks of mania may not be preceded by the usual prodromal symptoms that occur before the first attack. Temporary maniacal frenzy, epileptic mania, and explosive maniacal attacks in paretic dementia come on suddenly. The first is extremely rare, and is attended by symptoms quite characteristic of the disorder; the second is associated with epilepsy; and the third is only one feature of a chronic and distinct form of insanity. Great mental or physical shock may be the apparent cause of a suddenly developed mania. The simulator overacts the symptoms with which he is acquainted of the form of insanity that he tries to feign, leaves out other symptoms that should be present, and adds some that have no place in the particular form of insanity which he endeavors to simulate. A person feigning insanity usually makes the symptoms sufficiently obtrusive to attract attention when he is observed, and may be quiet when unobserved. The simulator does not, as a rule, endeavor to prove his sanity, but in some cases when this

has been attempted it has been done in so absurd a manner as to be strong evidence of sanity. An insane person who is depressed and suspicious or the subject of delusions of persecution frequently regards his relatives and best friends as his enemies. This is not invariably the case, however. The incoherent raving maniac manifests somatic signs of his disorder that cannot be simulated. In the milder cases the expressed ideas in the delusions and the surrounding circumstances bear a certain relation to each other. The ignorant almost always feign some amnesia and a degree of dementia with all forms of insanity that they attempt to simulate. Such symptoms are foreign to ordinary mania, melancholia, and paranoia. Complete amnesia is rare and limited to periods of great excitement in the simple forms of insanity, to epilepsy, alcoholic insanity, mental disturbance from blows to the head, and to the various forms of dementia. Simulators rarely present elaborate and systematized delusions, such as are found in paranoia.

The ruse of casually remarking to some one in the hearing of the subject after the examination is completed that if certain symptoms were present there would be no doubt of the existence of insanity, rarely succeeds except with the ignorant. With this class such a suggestion may result in the presence of the absent symptoms on the physician's next visit. If the subject whose mental condition is under investigation is weak-minded, the feigning of symptoms suggested by the physician would not prove the sanity of the individual. It is not infrequent in large asylums for the insane to find some of the weak-minded lunatics assuming the delusions and developing some of the symptoms of the less feeble-minded insane.

The simulator is worse when under examination and when in the presence of those whom he desires to impress with the genuineness of his insanity.

No rules can be given by following which, in the examination of a person alleged to be insane, feigned insanity can invariably be detected. Each case forms a subject for special study and investigation. An experienced, skilled, and well-trained observer will rarely fail to detect simulation if sufficient time is allowed for a thorough study of the case. The previous character of the subject should be ascertained. If there is a possible motive for feigning, this should not be ignored. A thorough examination into the physical and mental condition of the subject should be made. If he will write, it will often facilitate matters by getting him to write his history and all the circumstances connected with his arrest, indictment, and imprisonment. Frequently repeated visits by the examiner are necessary. Sometimes prolonged observation of the simulator may enable one to detect feigning. Under such circumstances a shrewd attendant who will watch the subject when he thinks he is not observed, and who will, from time to time, after he has succeeded in obtaining his confidence, engage him in conversation, can best play the detective. The physician will best succeed in his investigation by making himself and the object of his visits known. *He should not act the part of a spy.*

Mania.	Feigned Mania.
1. Gradual in onset, with a prodromal period of depression.[1]	1. Sudden in its onset.
2. If a crime is committed, the insanity is exhibited before the commission of such an act.	2. If a crime is committed, the insanity is not assumed until after its commission, and usually not until the subject is arrested and indicted.
3. If the patient is wildly incoherent, there are pronounced somatic signs of such a mental state.	3. No somatic signs except those that attend forced physical exertion.
4. In the milder forms there is a relation between the expressed ideas and surrounding circumstances.	4. No constant relation between the expressed ideas and the surroundings of the subject.
5. The excited mental state and muscular restlessness may continue for days, with only short periods of comparative quietness, and no sleep may be obtained during this time either night or day.	5. Becomes exhausted and falls to sleep at night. In fact, the simulator often sleeps soundly all night.
6. The facial expression and the mental exaltation correspond; there is no dementia, and the memory in the quiet periods is found to be quite good, with rapid and irregular speech.	6. The facial expression and the feigned mental exaltation do not correspond, and therefore give to the simulated psychosis an appearance of unreality; a partial demented condition is assumed, and memory in the quiet periods appears poor, with slow and measured speech.

Melancholia.	Feigned Melancholia.
1. Gradual (weeks or months) in its onset. Crimes are rarely committed by melancholiacs, but when they are, the insanity had previously existed.	1. Sudden in its onset. A rather frequent form of insanity feigned by the criminal, but the symptoms had not existed previous to the commission of the criminal act.
2. In the violent and agitated forms somatic signs are prominent.	2. No somatic signs.
3. In the quiet forms loss of flesh usually occurs and memory is preserved; no dementia.	3. The simulator often puts on flesh in prison; loss of memory and dementia often feigned.
4. The friends and relatives are often regarded as his enemies.	4. Shows no disposition to treat friends and relatives as enemies.
5. Facial expression is painfully sad.	5. Facial expression normal.

Temporary Maniacal Frenzy.	Feigned Temporary Maniacal Frenzy.
1. The outbreak is sudden, but it is preceded by a period of mental or physical depression.	1. The outbreak is sudden, and no evidence of mental disturbance is observed until after the criminal act is committed.
2. The patient is in a condition of maniacal excitement or melancholiac frenzy, and is destructive, violent, and incoherent usually for a few hours, when he falls into a deep sleep.	2. The subject frequently makes an attempt to escape, and often talks to the authorities if arrested a few minutes or an hour or so after the crime has been committed; is not violently incoherent immediately after the commission of the crime, and does not pass into a deep, stuporous sleep.

[1] Mania may begin comparatively suddenly in those who have suffered from a previous attack of this disorder, or when it is caused by some profound mental or physical shock.

3. On awakening, he is rational, but feels exhausted. The mind is clear, remembers everything up to the time when he became incoherent, but nothing more.	3. Frequently feigns to have forgotten the altercation or any of his actions and those of his victim immediately preceding the criminal act.

Paretic Dementia.	Feigned Paretic Dementia.
1. Developed gradually.	1. Assumed after the offense has been committed.
2. The speech defect and the moral and mental failure are so characteristic that this form of insanity is rarely attempted to be feigned by the ignorant.	2. The eye symptoms cannot be feigned, but these may be absent. Unless the simulator is an expert in insanity he will be detected if he is requested to read a manuscript containing the words "truly rural," "national intelligencer," "artillery," "stethoscope," etc.

Paranoia.	Feigned Paranoia.
1. Delusions elaborately systematized.	1. Delusions maintained by a very poor process of reasoning, even if the premises were correct.
2. Is quiet and frequently unobtrusive in regard to his delusions if not contradicted.	2. Seeks to make his delusions prominent, so that they will attract attention.
3. If his delusions are denied and ridiculed, he becomes angry and sometimes violent.	3. Bears opposition and even ridicule without becoming demonstrative.
4. Is not cautious in his speech, especially if excited.	4. Shows a disposition to weigh the effect of his words.

Amnesia is found in epilepsy, dementia, alcoholism, and may follow traumatism to the head, and during this mental state apparently intelligent acts may be performed of which the subject has only an indistinct memory afterward, or all knowledge of them may be entirely obliterated.

Amnesia.	Feigned Amnesia.
1. In amnesia from all forms of dementia recent events are less readily recalled than those of a former date, especially those in connection with the subject's childhood and youth.	1. Considerable amnesia is assumed for all periods of life, and the recent occurrences may be remembered and those of youth forgotten. May feign to remember nothing that occurred at the time of the commission of the act for which he is arrested, but is able to repeat the conversations he has had in his cell from day to day.
2. In all forms of temporary incomplete amnesia the things that are remembered will be those that would naturally have made the most impression on the subject's mind at the time of their occurrence. If a murder has been committed, the quarrel, the fatal shot, or some similar circumstance may be remembered, but the minor details will be forgotten.	2. The trivial things are more likely to be recalled, and all that relates to the subject's excited state leading to the commission of the crime will be forgotten.

It is rare for temporary amnesia to be complete unless caused by epilepsy, a blow to the head, or some decided emotional or physical shock. Most of these excited periods are followed by a stuporous sleep. Persons who commit crimes during periods of excitement, speak about them intelligently immediately afterward, and subsequently pretend to have forgotten everything connected with the crime are usually feigning. Some epileptics may commit crimes during their peculiar psychic conditions that occur between the convulsive attacks. Under such circumstances the history of the patient, his former mental aberrated states, and his actions during these must be carefully studied in each individual case.

Attempts at feigning a delirious state are usually unsuccessful, as the impostor associates it with a condition of dementia instead of a dreamy confusion.

Feigned Epilepsy.—When epilepsy is simulated by the hysteric for the purpose of gaining sympathy, or by the impostor in order to obtain alms or to secure care and attention in a hospital, the usual tests found in the books will generally suffice to detect feigning. When, however, the simulator is an expert scoundrel and assumes epilepsy in the hope of being excused from duty in the army or of being considered irresponsible if he has committed a crime, it is not always easy to distinguish the real from the feigned disease. Gowers[1] states: "The simulation of an epileptic fit is, on the whole, rare, and the pretended fit never closely resembles a genuine seizure. In all cases the perfect reaction of the pupil to light throughout the fit will be conclusive evidence." I am reluctant to differ from an authority so eminent as the one just quoted, but a careful study of cases of feigned epilepsy by criminals has convinced me that it is possible for some simulators to imitate fits that are not easily distinguished from the genuine. The results of a most careful study of feigned epilepsy will be found in an article on "Malingering" by Keen, Mitchell, and Morehouse.[2] This paper, prepared conjointly by three skilled observers from their own observations on soldiers during the late war of the Rebellion deserves more attention than it seems heretofore to have attracted.

The epileptic, in going into a convulsion, with or without a cry, falls unconscious, often striking his face, or he may suddenly sink to the ground to save himself if he has any warning of an approaching fit. The face is at first pale, but the convulsive movements, tonic in character, immediately follow the fall, the limbs and trunk muscles are fixed and rigid, respiration is arrested for a few seconds, during which the face first becomes flushed, then livid. As soon as the muscular rigidity begins to relax, clonic convulsive movements take place, and the patient breathes irregularly. The muscular disturbance is often more marked on one side of the body than on the other, and the head is frequently drawn downward and to one side by the irregular action of the sternocleidomastoid muscles. During the clonic convulsive move-

[1] *Diseases of the Nervous System*, second edition, vol. ii., p. 760.

[2] *American Journal of the Medical Sciences*, October, 1864, pp. 384–394.

ments the tongue is often bitten, and froth and mucus, tinged with blood, may ooze from the mouth, and sometimes from the nose. As the clonic convulsive movements become less frequent, the livid condition of the face lessens. After the fit, which usually lasts from one to three minutes, the patient may immediately pass into a stuporous sleep, or he opens his eyes and looks around in a dazed manner, may answer questions, but soon falls into a deep sleep; he may perform various automatic acts, or he may become maniacal. Personally, I have never witnessed the last as a termination of an epileptic fit. During the seizure the eyes may be turned or jerked in various directions, the pupils are dilated, and usually the irides are irresponsive to light; the patient is totally unconscious and does not feel pain, but the corneal and other reflexes are not completely abolished.

Everything usually observed in an epileptic fit may be simulated by a clever malingerer except the change in the color of the face, the condition of the pupils, and the insensibility to suddenly inflicted and unexpected irritation.

Color of the Face.—A simulator, by muscular action, can produce redness of the face, and by holding his breath this color will change to a darker hue. It is very difficult voluntarily to cease breathing until the face becomes deeply cyanosed, as occurs in genuine convulsions. It is rare for a simulator to be seen sufficiently early for absence of the paleness of the face to be noted.

Condition of the Pupils.—In the early stage of the fit the pupils are said to be occasionally contracted. If this phenomenon is present, it must be very evanescent in character, as I have never succeeded in exposing the eyes sufficiently early to find the pupils contracted. At all events, during most of the tonic stage and throughout the clonic the pupils are dilated, and the irides will not often respond perceptibly to light. As consciousness begins to return the extreme dilatation passes away, and the pupils may alternately contract and dilate every few seconds.[1] Personally, I have not seen a genuine epileptic convulsion during the height of which the irides responded to light.

Keen, Mitchell, and Morehouse, in the article on malingering previously referred to, state: "We ourselves are of the opinion that when, in a fit, the pupils, largely dilated, remain impassive and motionless before a bright light, the case is almost certainly a real one. But, unfortunately, this state of things is of very rare occurrence, even in severe fits. The pupils unquestionably contract in the presence of a bright light in many such cases. Sometimes the movement is sluggish and slight; in others, it is almost normal as to range and speed of movement." These observers experimentally demonstrated that violent muscular action will cause the pupils to dilate rapidly, and that so long as the muscular action is kept up, the irides will move only slightly and sluggishly to a bright light.[2] From their observations they deduce the conclusion that only in those cases of epileptic fits in which the

[1] Gowers states that this phenomenon was first pointed out by Reynolds.
[2] I have verified the accuracy of this observation.

irides fail absolutely to respond to light is the pupillary test of the genuineness of the convulsion of any value whatever.

Insensibility to Suddenly Inflicted Irritation.—In the unconsciousness from an epileptic convulsion, if the cornea is touched with the end of the finger, the eyelids may move a little; if the supra-orbital nerves are firmly pressed upon with the thumbs, the skin of the forehead may slightly corrugate; or if a pin be thrust into a limb, it may be flexed, but no purposive movements will be made to get rid of the irritant. In feigned epilepsy, if suddenly and unexpectedly acute pain be caused, an intelligent effort will at first be made by the simulator to avoid the irritant.

The test of a genuine fit which Keen, Mitchell, and Morehouse regarded of the most value is etherization. According to these observers, if ether is administered to a patient in an epileptic convulsion the violence of the muscular spasm is at first increased, but the subject passes into a profound comatose state without showing any of the cerebral excitement witnessed ordinarily in the administration of ether. Further, when these subjects are recovering from the effect of the anesthetic, they may have convulsive movements before complete consciousness returns. In feigned epilepsy the subject will laugh and talk during the cerebral excitement caused by the administration of ether, just as is generally observed. These writers add:

"When ether is used during the state of comatoid sleep which follows many fits, there is also an absence of all manifestations of excitement, and the sleep only becomes more intense.

"It appears to us that the ether-test as proposed by us is the most valuable and certain of all the means hitherto employed to unmask cases of feigned epilepsy."

The ether-test for determining the genuineness of an apparent epileptic fit would be more valuable and reliable if all persons who are not subject to epileptic attacks manifested unequivocal evidences of cerebral excitement before passing into a deep ether coma. It is not an uncommon experience, however, to have patients become deeply narcotized from ether without laughing or talking or manifesting very decided signs of cerebral excitement.

We have seen that the majority who attempt to feign a fit do it so bunglingly that the fraud is easily detected. It is only the expert simulator whose cleverness at deception often puzzles the most skilled physician. There is probably no one means by which feigning of a fit can invariably be exposed. What, then, should be the rule of procedure in cases where feigning is suspected, but cannot positively be detected by the ordinary means resorted to for this purpose?

"We should observe, first, the patient's face and mental condition, to see if they have been influenced as yet by the disease. Next, we should obtain his personal character as nearly as possible, and all the particulars of the origin of his malady and of his general health, to see if there were any contraindications developed. We should observe where and how he was attacked by the fit. One of our patients, whom

we had suspected, set our suspicions at rest when we learned that he unquestionably had a fit in his sleep.[1] We should then observe one or more of the attacks, and never should we decide without doing so. If we can discover any true signs of real consciousness or sensation, unquestionably the man must be a malingerer. Hence, if he grows worse when visitors are present; if he opens his eyes, evidently to see what is going on; if he does not fall off the bed when not held, but struggles sufficiently to do so when he is held; or if he exhibits pain, not reflex movements from irritants purposely applied or from self-inflicted injuries; or if he should suddenly recover when severe measures are proposed in his hearing—we should class him as a malingerer. Perfectly immobile pupils, especially in the absence of violent spasms, we should regard as an evidence of real epilepsy, but only when they were thoroughly and carefully observed during the spasm itself, which is often impossible. A genuine epileptic will usually keep his hands open if they are forcibly unlocked, while a malingerer rarely does so."[2]

Severe pressure with the thumbs on the supra-orbital nerves during a genuine spasm never causes any purposive movements; simply a slight corrugation of the skin of the forehead. So much pain may be caused by this method that it is difficult for a simulator to prevent attempting, at least, purposive movements directed toward getting rid of his tormentor. I have never met with a case of feigning in which this test has completely failed. Irritation of the cornea is hard to bear by the simulator without showing some signs of pain. Very few simulators can bear, without giving some expression of suffering, a strong galvanic or faradic current applied to the dry and powdered skin by means of a dry wire-brush, especially if they do not know beforehand that the skin is going to be severely irritated. Various states of feigned unconsciousness may be exposed in this manner.

Since accident-insurance policies are so commonly carried by all classes of persons, feigned and exaggerated disease and suffering have become common. The ease with which jurors, and often physicians, are deceived by the actions and statements of persons who have sustained only slight injuries is another incentive for the dishonest to magnify their suffering or to simulate some disease the existence of which would entitle them to liberal compensation.

Feigned Headache.—Of all symptoms of disease, headache is the easiest to feign, and its existence may be impossible to disprove when this symptom is assumed by a clever simulator. In the alleged presence of this symptom, as in that of any other of the nervous system, all the organs of the body should be subjected to a most thorough and searching examination. In the absence of an apparent cause it does not prove that the headache is feigned. We all know how frequent it is for the physician to meet with persistent and distressing headache in persons in whom there can be no suspicion of feigning, and

[1] Too much stress must not be laid on the occurrence of a fit during sleep, as the latter may be feigned.

[2] Keen, Mitchell, and Morehouse, *Amer. Jour. Med. Sci.*, October, 1864, p. 389.

yet no probable cause for the cephalalgia is detected. The character and seat of a feigned headache may often be changed at will by suggestions skilfully made to others in the hearing of the simulator. Failing this, I know of no means by which one can say with positiveness that no headache exists. On having the patient watched, and finding that he acts differently when he thinks he is not observed by the physician, fraud may be suspected.

Vertigo, when feigned, is equally as difficult to detect as is headache.

Pain and Tenderness.—These may be complained of in any portion of the body. The most common sites for feigned pain are in the spine, back, hips, or legs. Real pain in the spine will be attended with some rigidity of the spinal muscles, and the gait and carriage of the patient will be in keeping with the severity of the pain, and will not be changed as soon as the patient thinks he is not under observation. Pain in the nerves of the legs or radiating from the spine down the nerves will follow the course of these nerves. Simulated tenderness may often be detected by pressing on the alleged tender spot while the patient's attention is absorbed in some subject of conversation. It must be remembered that real pain in the spine and nerves often exists, and is exaggerated by the patient's attention being concentrated on his suffering. Great care is always necessary, lest by a superficial examination and a hasty conclusion injustice to an actual sufferer is done. Many mistakes of this kind have been made by men of experience and skill. It may be accepted that the greater the examiner's knowledge of the functions and diseases of the parts, and the greater the thoroughness and patience displayed in the examination, the less frequently will real disease go undetected and the less the chances of the simulator will be of escaping exposure.[1]

Paralysis of motion may involve one limb or part of a limb, one entire side of the body, both legs, rarely both arms, or nearly all the voluntary muscles may be affected. The sphincters of the bladder and rectum and nearly all the muscles supplied by the cranial nerves may be paralyzed. The paralysis may be organic, feigned, or hysteric. The first question to decide is whether the loss of power is due to organic changes in the muscles or nervous system. If it is due to disease of the muscles, it is either acute or chronic. The acute variety is attended by inflammation, tenderness, pain, and swelling in the affected muscles. The chronic form is attended by wasting of groups of muscles—progressive muscular atrophy of the idiopathic variety. This cannot be successfully feigned.

A lesion in the nerves, cord, or brain may give rise to paralysis.

[1] The so-called "Mannkopf plan" may be utilized: If pain and tenderness are real, pressure upon the painful parts increases the rapidity of the heart's action. In simulation the pulse would not change. This scheme is not to be wholly trusted, however, as in exceptional instances an increase of real pain is not followed by a more frequent pulse; while, on the contrary, the pulse may increase during the experiment in a case of simulation.—EDITOR.

If the paralysis is caused by trouble in one nerve or a plexus of nerves, the loss of motion will be limited to the muscles supplied by these nerves. The character of the paralysis, the changes in sensation, the trophic disturbances, and the electric reactions will suffice to determine whether the loss of motion is due to disease of the nerves. Multiple neuritis is bilateral, usually symmetric, affects the parts most at the distal portions of the limbs, is attended commonly by sensory and motor defects, alterations from the normal in the reflexes, trophic disturbances, and electric changes. If the paralysis is due to a lesion in the anterior horns of the cord, there will be no pronounced sensory disturbance, the muscles will be flaccid, atrophy early, and present electric changes. If a group of muscles only is involved, it will be those concerned in the performance of definite associated movements, instead of all those supplied by individual nerves. In paralysis from myelitis, both motion and sensation will be, to a greater or less degree, affected from a point on a level with the height of the spinal lesion downward. If the lesion is in the lumbar region, the sphincters of the bladder and rectum will be paralyzed and the urine will dribble away as it reaches the bladder. An examination of the rectum by the finger will readily detect the paralysis of the rectal sphincter. Muscular wasting will occur, electric changes may be detected, a sacral bed-sore will probably form, and the deep reflexes will be abolished. If the spinal-cord lesion is above the lumbar region and sufficient to paralyze the parts innervated by nerves given off below the seat of the myelitis, the reflexes on a level with the lesion will be abolished, the deep reflexes in the parts below will be increased, there will be little tendency to the formation of bed-sores if cleanliness is maintained, electric and trophic changes in the parts below the level of the lesion will be slight, and while voluntary control of the bladder and bowel is lost, the sphincters are not paralyzed. If the urine dribbles, it is because the bladder is full and the incontinence is that of overflow. No voluntary effort can be made to evacuate the bladder and bowel. Spinal paralysis, except that form due to disease of the anterior horns (poliomyelitis), is generally bilateral.

Cerebral paralysis of organic origin is, except in very rare cases, unilateral in character. The most common form is the hemiplegic—the leg, the arm, and lower side of the face on the same side. In a number of instances the paralysis is monoplegic at first, the face, arm, or leg being affected. In a still smaller number of cases the paralysis is "crossed," the face is paralyzed on one side, and the leg and arm on the opposite side. In paralysis from organic cerebral disease the deep reflexes are often increased on both sides of the body, but to a much greater extent on the affected side. Sensory disturbance may be present or absent, depending upon the seat of the brain-lesion. Electric changes are usually absent. The greater the tendency to contractures, the more pronounced the trophic changes in the paralyzed parts.

Disturbance of sensation due to organic trouble will, when caused by lesion of one or more nerves, follow the cutaneous distribution of the nerves, and be associated with some loss of motion unless a purely

sensory cranial nerve is affected. If the loss of sensation is from a lesion of the cord or brain, the attendant symptoms will enable a diagnosis to be made.

If paralysis exists and no evidence of an organic lesion is found, it is not always an easy matter to make a sharp distinction between feigned and hysteric paralysis, because few cases of hysteria are encountered in which the symptoms are not exaggerated, and some may be feigned.

Space will not permit a thorough discussion of hysteric paralysis. Quite frequently the loss of motion is associated with loss of sensation. If the anesthesia is limited to the limbs, it involves the limbs up to its junction with the body, where it abruptly ends. It thus differs in its distribution from anesthesia due to an organic lesion. When the loss of sensation affects one side of the body, it involves the face, scalp, and special senses on that side. There is a condition of "crossed amblyopia," differing from the hemianopsia of organic origin. I have never met with a case of "crossed amblyopia" of organic origin. The reflexes are increased in hysteric paralysis, but they are usually alike on both sides of the body. In hysteric paralysis, if the patient is requested to make an attempt to contract the flexor muscles of a limb, the extensors may be felt to contract at the same time. This does not occur in paralysis from organic origin. Further, hysteric paralysis is often changeable. The history of the patient will usually reveal former attacks of a hysteric nature.

Feigned paralysis presents no symptoms of an organic nature, and, on account of the ignorance of the simulator, the symptoms are greatly exaggerated and there is an absence of those stigmata so commonly found in pure hysteria. If anesthesia is feigned, it may be detected by the electric brush applied when the simulator is not expecting it. If the subject is blindfolded and a feather or camel's-hair pencil used to mark out the area of anesthesia, the latter will be found to vary considerably from minute to minute, as it is impossible to feign cutaneous anesthesia that ends abruptly. The methods employed to detect feigning in general may be resorted to with advantage in the examination of suspected feigned paralysis. Some movements may take place in the paralyzed limbs from organic disease if the patient is anesthetized, but the muscular movements are purely reflex, while in feigned paralysis the movements will be equally vigorous on the two sides of the body. Hutchinson[1] has shown that a healthy limb trembles when a heavy weight is suspended from it.[2]

Feigned contractures are sometimes most difficult to detect.

[1] Hamilton, *Medical Jurisprudence*, p. 211.

[2] The so-called "Babinski reflex" may be tried: In a normal adult, when the sole of the foot is tickled, the great toe bends downward or is flexed. In an organic paralysis of one or both legs, the great toe is extended or bent upward. In a hysteric paralysis of one or both legs, and in simulated paralysis (unless simulated by one cognizant of this sign), irritation of the sole of the foot would produce the same movement of the toes as in normal conditions. There are exceptions to the rule thus laid down, but the Babinski test is valuable in some 80 per cent. of all tests.—EDITOR.

They should be investigated in the same manner as is pursued in examining a case of paralysis. A thorough examination will fail to reveal any evidence of organic disease.

If the feigning is continued long enough, the contractured muscles will shorten, and some wasting of the parts will take place. Before organic changes have occurred in the muscles, if the patient is surprised in his sleep, the contractured muscles will be found relaxed and the limbs may be suddenly extended before the simulator recovers consciousness sufficiently to resist extension of the limb. A clever rogue may simulate paralysis or contraction of a limb so skilfully as to almost defy detection. Complete relaxation under an anesthetic of a limb that has been contractured for months would be in favor of feigning.

Catalepsy is extremely rare and difficult to feign. The muscles are in a wax-like condition, and gradually yield to gravity when the limb is extended at right angles with the body. If a weight is suspended to the limb and the latter is held rigidly in the same position, the subject is probably feigning.

Sleep may be simulated. In sleep the pupils are very small, and on awakening the subject and raising his eyelids, they will dilate at first, even in the presence of a bright light. A person may voluntarily contract his pupils by a strong effort at convergence of the eyes. Excluding all diseases or drugs that contract the pupil, pin-hole pupils without convergence of the eye would be in favor of the genuineness of the sleeping condition of the subject.

Feigned unconsciousness may usually be detected by using some irritating agent—the faradic brush suddenly applied to the skin when the simulator is not suspecting anything of the kind and is totally unprepared to nerve himself against pain will cause purposive movements. It is probable that the method employed and recommended by Keen, Mitchell, and Morehouse during the war of the Rebellion to determine the genuineness of epilepsy may be used in detecting feigned unconsciousness in general.[1] These observers found, on anesthetizing a person who was feigning unconsciousness from a simulated fit, that he would invariably struggle during the stage of cerebral excitement, but no evidence of such excitement was ever manifest if the assumed unconscious state was real.

Tetanus, chorea, the systemic diseases of the spinal cord or **pronounced disease of the brain** could not be successfully feigned, even by a clever simulator, if the medical examiner investigates the condition critically in the light of a detailed history.

Aphonia may be feigned for an indefinite period. The deception can be detected by placing the simulator under an anesthetic, preferably ether. During the stage of cerebral excitement, as the will no longer restrains the action of his vocal muscles, he talks quite distinctly. I am not aware of any reliable method for distinguishing hysteric from feigned aphonia, aside from the history and general hysteric stigmata of the former.

[1] *Amer. Jour. Med. Sci.*, October, 1896.

Sciatica.—Under the heading pain and tenderness a few remarks are made on feigning, but as sciatica is commonly assumed by impostors, some of the principal points in the diagnosis may be mentioned. There is pain along the course of the nerve, and often along that of its branches, and not infrequently in the areas of the final distribution of the nerve. The pain is increased by putting the limb in such a position as to make the nerve tense; and the knee is kept slightly bent in walking so as to prevent any increased tension of the nerve. Gowers gives seven spots of tenderness: (1) Above the hip-joint, near the posterior iliac spine; (2) at the sciatic notch; (3) about the middle of the thigh; (4) behind the knee; (5) below the head of the fibula; (6) behind the external malleolus; (7) on the back of the foot. It is scarcely necessary to say that all these points of tenderness are not present in every case. The patient, in pointing out the course of the pain, maps out the distribution of the branches of the nerve. The main nerve-trunk at the back of the thigh is often extremely tender to pressure. If the leg is extended at the knee and the thigh flexed upon the pelvis, the patient will experience pain at the sciatic notch, along the course of the nerve at the back of the thigh, at the back of the knee, and often in the heel. In feigned sciatica the pain is not definitely located along the course of the nerve and at the points of its distribution. In feigned cases the points of tenderness are not always over the body of the nerve or branches of distribution. Pain on the inner side of the thigh is not caused by disease of the sciatic nerve. It should be borne in mind that when the sciatica is due to pressure or irritation within the pelvis there may be no points of tenderness along the course of the nerve, but the pain experienced by the patient will be referred along the course of the nerve, that of its branches, and more especially to the points of distribution of the nerve.

Rheumatism.—DaCosta says: Chronic rheumatism is often feigned, especially by malingerers in the army and navy, and the deception may be difficult of detection. They pretend to be scarcely able to walk or hobble around with a cane, and complain much of pain and stiffness in their joints. Yet there is not the least sign of deformity or real stiffness; the pain is always stated to be the same, and their general health is excellent. Their way of using the stick, too, is characteristic; they move it each time they move the seemingly crippled leg, but, as a rule, not immediately, thus not employing it as a support. Anesthetics are of great value in enabling us to decide as to the real amount of immovability of the limb."[1]

Hysteria is sometimes feigned for the purpose of gaining sympathy. It is not always easy to determine whether the symptoms in a case of assumed hysteria are genuine or simulated. The deception is made all the more difficult from the fact that many hysteric subjects feign some of the symptoms. If the history of the case gives no former attacks or symptoms of hysteria and the general stigmata of the disease are absent, it is fair to presume that the subject is a malingerer.

[1] DaCosta, *Medical Diagnosis*, seventh edition, p. 812.

Vomiting is not infrequently simulated by malingerers. Some persons learn to vomit apparently at will; others induce nausea and vomiting by irritation of the fauces. A short time ago I had a man under my care who had been injured in a mine. He had a suit pending for large damages. It was reported to me by the nurses in the hospital that he vomited about an hour after each meal, and between meals he vomited almost immediately after drinking any liquid substance. I expressed great concern in the presence of the patient about the gravity of his case, and requested him to drink a glass of water in my presence that I might witness his distressing symptom and be able to testify in regard to his condition. He hesitated at first, ostensibly on account of the distress that he experienced on imbibing any liquids. I was persistent, and insisted that I must observe the vomiting. He drank a glassful, about eight ounces, of water, and almost immediately I noticed that he began to retch. On placing my hand on the abdomen I found that he was voluntarily contracting the abdominal muscles and the diaphragm. These movements continued every few seconds for about two minutes, when he succeeded in ejecting from the stomach nearly all the liquid he had drank. The emesis occurred without any pallor of the face, increased flow of saliva, or any other evidence, so far as I could determine, of nausea. I at once charged him with malingering and of voluntarily inducing the emesis. I threatened that if he vomited any more I would testify against him when his case came to trial. He did not vomit again, either liquids or solids, although he remained under my observation several weeks longer. In cases of suspected feigned vomiting the patient should be watched carefully, when the deception will ordinarily be readily detected.

Hematemesis.—There are several ways by which a clever simulator may cause vomiting of blood. The mucous membrane of the nose, mouth, or fauces may be irritated until it bleeds and the blood be swallowed, or blood may be obtained from other sources and taken into the stomach. In suspected cases of fraud the supposed malingerer should be watched carefully and the exposed air-passages examined.

Suppression of Urine.—The man who feigned vomiting, an account of whose case is given above, simulated a partial suppression of urine before he learned to vomit at will. The nurse reported that he apparently passed only about three ounces of urine in twenty-four hours. I requested that the entire quantity passed during this time should be saved in a vessel for me. The quantity was three and one-half ounces. It was normal in color, slightly acid in reaction, and had a specific gravity of 1010, and contained neither albumin nor sugar. It was quite evident from the analysis of the urine that the man was passing more than this quantity of water. His bowels had moved only once during the twenty-four hours. I asked him if he had not passed more than three ounces and a half of urine. He declared that he had not. I left orders with the resident physician that the patient should be catheterized every four hours during the next twenty-four hours. The result was about the same—only four ounces of urine were obtained.

This specimen was normal in every respect. I left instructions that the catheter be employed in the same manner during the next twenty-four hours; at the same time ordered the patient to be closely watched, and the entire quantity of liquid drank to be measured without his suspecting what was being done. Again the quantity of urine passed and drawn off by the catheter was only three and a half ounces. No one had seen him go to the water-closet or any other place in which he might urinate. It was certain that he had not gone to the toilet-room during the day. He was undressed and apparently in bed all the time. The quantity of liquid that he had drank during the twenty-four hours that he had been under such close observation was 48 ounces. It was reported that he had not perspired much. His temperature, pulse, and respiration were normal. During the time that this man was under observation in St. Luke's Hospital I had been conducting a series of observations on afebrile patients in the Arapahoe County Hospital to determine the relative quantity of urine passed to the quantity of liquid ingested. In no case, while the patients were in bed, had the quantity passed been less than two-thirds of the quantity ingested, and in most of the cases the former quantity was about three-fourths as much as the latter. Thus fortified, I boldly charged the man with lying, telling him that I had measured every drop of fluid that he had drank, and that there could be no such discrepancy. He confessed. During the day he had kept a vessel secreted back of the wardrobe, into which he had urinated, and between midnight and day, when everything was still, he had quietly gone to the water-closet and emptied the vessel.

Diarrhea and constipation are sometimes feigned, but the fraud may easily be detected by having the malingerer closely watched.

Blood-spitting or **hemoptysis,** if assumed, can usually be detected by a careful inspection of the mucous membrane of the nose, mouth, and fauces.

Feigned asthma, according to DaCosta, is not worse at night nor much increased by exercise.

Feigned Poisoning.—An evilly disposed person might assume some of the symptoms produced by a poison and accuse an innocent person, against whom the simulator had a grudge, of administering the poison. The symptoms would be ill feigned by an ignorant person. One well versed in the action of poisons might take a cathartic and an emetic and then place a poison in the discharge from the bladder and bowels or in the vomited matter. The finding of a poison in these would be no proof that it had come from the body of the person alleged to have been poisoned, unless they had been passed into clean vessels in the presence of a physician or some other reliable witness. If the urine drawn with a catheter showed the presence of the poison, this would be conclusive evidence of its having been within the patient's body, and nothing more.

Feigned Pregnancy.—Pregnancy may be feigned to extort charity, to secure an unjust settlement of property, or to escape infliction of condign punishment. The deception may be detected by skilled

physicians versed in the symptoms of pregnancy. In no case should an opinion be given without a careful internal and external examination of the alleged mother. If the period of gestation has not advanced sufficiently to permit of a definite opinion in regard to the woman's real condition, the physician should not hesitate to ask for an extension of time, during which repeated careful observations should be made.

Feigned Menstruation.—A woman may be pregnant, yet, desiring to conceal her condition, feign menstruation by soiling her linen with blood. The chemical tests are uncertain for distinguishing ordinary blood from that found in the menstrual flow. An examination of the woman to determine the presence of the results of conception will usually suffice to settle the question. It must be borne in mind that a few persons menstruate throughout the period of gestation. I have met with one woman who menstruated only during gestation and lactation. Throughout these periods her menstrual epochs occurred regularly every twenty-eight days. Suppressed menstruation could be determined only by keeping the suspected person under close observation.

Feigned Delivery.—Taylor says: "Delivery has often been feigned by women for the purpose of extorting charity, compelling marriage, or disinheriting parties who have claims to an estate, and in other cases without any assignable motive." In most of these cases it is recent delivery that is assumed, and an examination of the alleged mother by a competent medical man would soon determine the matter.

Feigned Abortion.—If a woman charges another person with having perpetrated the crime of abortion on her, the truth or falsity of the statement may be easily demonstrated by a physical examination, if this is made soon after the crime has been committed. If the charge is made several months after the alleged commission of the crime, it should raise a suspicion of deception on the part of the woman making the charge. At so late a date it may be impossible to decide positively whether an abortion has been performed.

Feigned Strangulation.—In the few cases of simulated strangulation the marks of violence around the neck have been slight or entirely absent, the impostors have retained sufficient consciousness to take cognizance of everything that has occurred, the cord around the neck has not been sufficiently tight seriously to endanger life, produce cyanosis of the face, protrusion of the tongue, or congestion of the eyes, and the other circumstances surrounding the alleged attempts at murder have been such as to prove the innocence of the accused parties.

If a serious, but unsuccessful, attempt is made to commit murder by strangulation, either by means of the hands or a cord tied around the neck, the parts would not be so delicately handled as to leave no marks of violence. If a cord were tied around the neck, it would be with a firm knot, and the constriction would be so great as to endanger life unless the cord were removed within a few minutes from the time it was applied. There would, in all probability, be evidence of a desperate struggle, and the general circumstances would be in keeping with the acts of the attempted murder.

THE LEGAL ASPECTS OF PREGNANCY.

It is exceedingly important that the medicolegal expert be well versed in the clinical signs and manifestations of the various stages of pregnancy, since it is not at all infrequent for the question of gestation or the effects of child-bearing to arise in a certain class of cases. Thus, in civil law a physician may be called upon to testify as to the virginity of a woman or the purity of a divorced wife libelously accused of gestation, as proved, negatively, by the absence of the signs of pregnancy or of parturition; he may be required to show the absence of pregnancy in cases of feigned gestation, or when a woman claims to be with child by a man recently dead, for the purpose of advancing an additional heir to a disputed title or estate, or for the purpose of substituting a child in place of the legitimate heir. The claim of pregnancy may be made in order to institute blackmail; it may be advanced as a plea to prevent attendance upon the witness-stand in an important trial by jury; it not infrequently is employed as a means of enforcing marriage to satisfy feelings of unrequited love or to establish a claim for financial support. It has been used as a means of holding the affections of an estranged husband desirous of offspring.

In the criminal court instances are recorded of women pleading pregnancy to bar execution. From the time of the old Roman law to the present such a claim, if verified, has been ample to postpone the carrying out of the sentence of capital punishment until the birth of the child is accomplished. In all such cases, under writ of *de ventre inspiciendo*, proof of the supposed condition is required, and is secured by careful investigation by medical experts or by the physician appointed by the court. These have rightfully supplanted the old English jury of twelve matrons who were summoned to ascertain the unfortunate woman's condition, and whose main duty was to determine whether or not the woman was quick with child. If the woman is acting *bona fide*, the examination will not be refused; indeed, it will probably be insisted upon by her. Refusal to permit the examination will expose her to the strong suspicion of unfair dealing. The practitioner must remember, however, that he becomes liable to legal action on her part if he force an examination against her will. It should be his duty, likewise, to warn the patient in all cases that any suspicious appearance may be used as valuable evidence against her. Again, the existence of pregnancy may be denied strenuously by a woman in order to avoid the stigma of illegitimacy, or to gain time for the induction of an abortion.

Here the effort of the physician should be to demonstrate the presence of the gestational signs.

It is important to note that a positive diagnosis of pregnancy is impossible before the sixth week, and often not until the second menstrual epoch, especially in multiparous women. After the date of the positive signs no difficulty should be experienced in proving or disproving the existence of pregnancy. Hence the examination should be postponed, if possible, until such a time as will elicit positive results. An error in diagnosis, whereby an innocent woman is erroneously declared by the examining physician to be pregnant, will react disastrously upon the so-called expert, and may prove ample ground for the institution of legal proceedings.

It is patent, therefore, that a knowledge of the signs of pregnancy is essential for a positive affirmation or denial in a given case.

THE SIGNS OF PREGNANCY.

It is customary to group the signs of pregnancy broadly under the two headings of *subjective* and *objective*, of which the latter only—those detected by the senses of the examining physician—are to be trusted implicitly, since they alone are capable of demonstration. While the subjective signs, when present, are exceedingly valuable as suggestive of pregnancy, it must be remembered that a woman may be voluntarily deceiving her physician, or that she may be herself deceived, as has been frequently noted in remarkable instances in the history of obstetrics. Thus, a woman nearing her menopause and married late in life, or sterile since an early marriage, may readily coax herself into believing that the menstrual suppression characteristic of the so-called "dodging-period" of the climacteric is due to an incipient gestation. Once firmly fixed in her mind, this belief can grow *pari passu* with the supposed advancing pregnancy. Intestinal movements will be interpreted as fetal motions; gaseous distention of the bowels and fat-accumulations in the abdominal walls are the progressively enlarging uterine body; the irritable bladder of elderly women is construed to mean the irritable bladder of uterine pressure; the breasts may be found to secrete a small amount of serous fluid, and the woman and her husband, and even the family doctor himself, may be deceived into believing that pregnancy exists. In other words, a typical *pseudocyesis* or *phantom pregnancy* is developing.

Let it be noted here that pseudocyesis, with the one exception of abdominal enlargement, is a subjective consciousness entirely. The woman feels the sensation of quickening, the fetal movements, the pressure upon the bladder, the sympathetic breasts. The diagnosis of her true condition can be made by the skilled obstetrician only by giving attention to the following points: The woman's age—she is approaching the menopausal period when menstrual suppression or irregularity is the rule, and nervous manifestations are frequently exaggerated; abdominal palpation and percussion will reveal a heavy accumulation

of fat in, together with a lax and pendulous condition of, the walls, and a highly resonant or tympanitic note over the entire surface; no fetal outlines can be detected by palpation of the abdominal surface; examination of the vulvar orifice and vagina reveals the senile atrophy and probably the beginning yellow discoloration of the mucous membrane that are characteristic of advancing years; the vagina may be more or less contracted, the cervix senile and atrophic, and the uterine body small and undergoing retrograde changes; the appendages are also small and detected with difficulty. In order to avoid any error in diagnosis it is better in these cases to administer chloroform or ether, when the abdominal enlargement will collapse and the true condition be made patent. These cases of genuine pseudocyesis may be much more difficult to diagnosticate than feigned pregnancy in hysteric women (see Feigned Pregnancy, p. 44).

It is well, then, to note that a pregnancy may be *presumptive*, it may be *probable*, or it may be *certain*. It is presumptive when, without any expert investigation, the woman, having been exposed, legitimately or illegitimately, to the possibility of conception, presents some or all of the subjective signs of pregnancy, together with a minor degree of abdominal enlargement. It is probable if these signs steadily increase in intensity and other signs develop at proper periods, which in a typical case of gestation should be superadded to the initial symptoms. Finally, a pregnancy is certain only when there are present the so-called positive or diagnostic signs of pregnancy, variously estimated as from three to five in number. They include the fetal heart-sounds, the fetal movements, ballottement, vaginal and vulvar discoloration, and intermittent uterine contractions.

For convenience of reference the signs of pregnancy may be grouped under four headings: namely, the uterine, the vaginal, the abdominal, and the general.

THE UTERINE SIGNS.

Cessation of Menstruation.—This is probably the most valuable of the subjective signs of pregnancy. If the previous history has been one of regularity, greater value is to be attributed to the irregularity or suppression of the function, especially if this be associated with other suspicious circumstances, as the history of sexual intercourse. It is the usual result of gestation, although it may follow other pathologic causes, or it may be absent altogether, and the woman menstruate regularly throughout the first trimester or even the full period of pregnancy. Other remarkable cases are those in which the normal (?) condition of the woman is that of amenorrhea, save during the progress of gestation, when regular menstrual discharges prevail. Again, it is possible for pregnancy to occur in young girls prior to the establishment of the menstrual flow, and for rapid successive pregnancies to prevent the appearance of the menses until late in the woman's life, or even altogether. Again, cases are on record of conception having occurred some time after the woman had passed through her climacteric. These

instances all go to prove that ovulation, the essential feminine process in conception, is not at all dependent upon menstruation, although it is probable that each menstrual epoch is associated with the escape of an ovum either at its height, shortly before, or shortly after its appearance. In other words, it is not necessary for a menstrual discharge to accompany each escape of an ovum.

Leopold and Mironoff[1] find that menstruation is usually accompanied by ovulation, but not rarely is unattended by ovulation. It is not dependent upon the maturation and rupture of a Graafian follicle, but the presence of the ovary and a sufficient development of the uterine mucosa are necessary. Ovulation is connected with menstruation in so far as it requires for its occurrence a congestion of the sexual organ lasting several days. Ovulation occurs independently of the time of menstruation, but under physiologic conditions, rarely. Usually menstruation occurs with ovulation; less frequently it may occur without ovulation; and least frequently ovulation may occur without any sign of blood at a regular menstrual period.

To still further complicate matters, there may be total absence of menstruation, the result of occlusion of some portion of the lower birth-canal (atresia of the cervix uteri, vagina, or vulva; imperforate hymen), with a vast accumulation of the fluid and great distention of the uterine and abdominal cavities, thus closely simulating an advanced pregnancy, with total suppression of menstruation. The persistence of menstruation during the first trimester of pregnancy, in addition to confounding the diagnosis, will be a confusing element in the determination of the duration of a given case.

The morbid conditions that are frequently associated with menstrual suppression without coincident gestation, and which are frequently overlooked by the examining physician, are incipient phthisis, chlorosis, anemia, hemorrhage from wounds or other mucous surfaces (**vicarious menstruation**), various forms of insanity, hysteria, sudden and decided change of climate, and the development of certain pelvic tumors as ovarian cysts, and occasionally uterine myomata. An important fact worth noting is that the menstrual suppression of pregnancy is generally associated with a steady improvement in the body-health, which is not true of the foregoing conditions. Anxiety, as after illicit intercourse, may temporarily cause menstrual suppression.

Feigned Menstruation.—Occasionally women will feign menstruation in order to conceal an illegitimate pregnancy. This is accomplished by staining the underclothing and napkins with other mammalian blood, which is not readily distinguishable from the menstrual flow; or even, as has been noted, with preserved menstrual blood. In the former case microscopic examination will generally show the absence of the typical vaginal epithelium, and such blood is much more predisposed to coagulation than are the true blood and acid vaginal discharge.

Progressive Increase in the Size of the Uterine Body.—This is a fairly presumptive sign of pregnancy after the first trimester,

[1] *Arch. f. Gyn.*, Bd. xlv., H. 31.

although not an infallible one. The tumor of pregnancy, at first ovoid in shape, becomes progressively more and more pear-shaped, occupies a median position with a slight inclination to the right, is smooth in contour and of a soft and elastic consistency, and is freely movable. It is of rapid growth, occupying fixed positions at certain periods of gestation (Fig. 1). Thus, at the second month, the fundus is about on a level with the top of the symphysis pubis; at the fourth month it is midway between this point and the umbilicus; at the sixth month it is at the um-

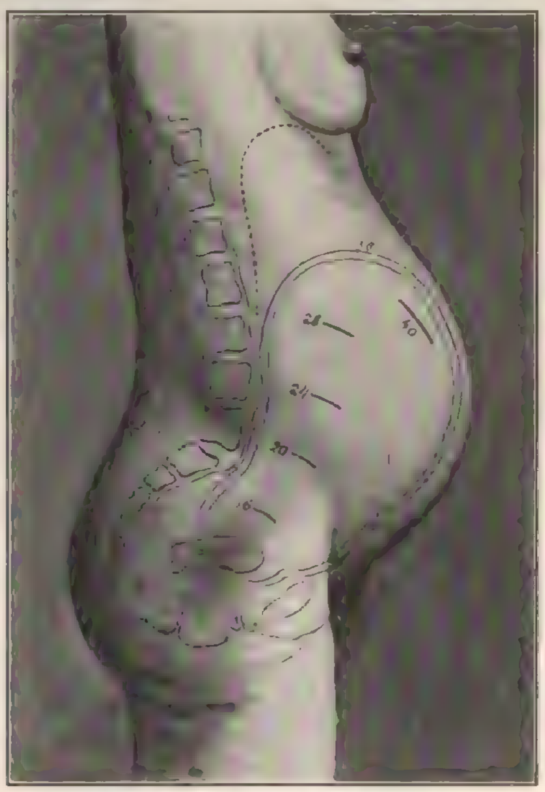


FIG. 1.—Abdominal enlargement of the sixth month of pregnancy, showing position of the fundus of the uterus at different weeks of gestation.

bilicus; at the seventh month it is midway between the umbilicus and the xiphoid cartilage; and at the ninth month it has fallen about one inch. The percussion-note over the uterine body is dull, due to the upward and backward displacement of the bowels. Other conditions that may simulate such a tumor at an early or later period are a subinvoluted uterus, retained menses forming a hematometra, a non-nodulated fibrous or myomatous tumor, gaseous distention of the bowel, ascites, an ovarian cyst centrally situated, and tumefactions of the abdominal organs, as the liver and spleen. It is very important that an

error be not made in this diagnosis, since many a woman has had her life embittered and the physician his professional reputation injured by such a mistake. Legal action can readily be grounded upon such an error. Intestinal flatus will be detected by the percussion-note ; ascites and ovarian cyst, by variation in the area of dulness and the elicitation of fluctuation ; the absence of correlated signs of pregnancy will distinguish other growths, while the uterine contractions may be noted in true pregnancy when the cold hand is laid upon the surface of the abdomen. Etherization in doubtful cases will be imperative.

Braxton Hick's Sign.—Intermittent uterine contractions capable of recognition after the third month of gestation must be regarded as an exceedingly valuable proof of gestation. No other known tumor than a uterine growth will present this phenomenon. At regular intervals from the fourth to the tenth month of gestation, varying from five to twenty minutes, the uterine tumor will harden and remain contracted for the space of from three-fourths to five minutes. This process is absolutely painless and is not recognized by the woman herself. The mere grasping of the uterus may cause it to appear. It is never absent in pregnancy, whether the fetus is living or dead.

The Uterine Souffle or Placental Bruit.—This is a very constant but non-diagnostic sign of pregnancy, since it may occasionally be detected in other uterine and ovarian tumors or conditions of marked pelvic congestion. It is a rhythmic blowing sound occurring synchronously with the woman's heart-beat, commencing about the tenth week of gestation, and persistent throughout ; it is situated low down and to one or the other side of the uterine tumor. It is produced by the rush of blood through the enlarged uterine arteries, and is generally most prominent in anemic individuals. It must not be mistaken for a similar sound, known as the **cardiac souffle,** which is produced by the passage of the blood through the foramen ovale.

The **funic or umbilical souffle** is a high-pitched, whistling sound synchronous with the fetal heart-beat, and heard best, although even then with difficulty, over the fetal chest. It is produced by tension of the cord with stenosis of its vessels, and has but slight value as a diagnostic sign of pregnancy. It is claimed that it is more readily heard when the cord is wrapped around the fetal body.

Irritability of the bladder is produced early in gestation, and in the closing month by direct pressure of the uterine body upon the base of the bladder. It is, in primiparous women, a sign of much value, especially if it be associated with a history of coitus and of menstrual suppression. It is by no means diagnostic.

VAGINAL SIGNS.

Jacquemin's Sign.—A bluish or purplish discoloration of the vagina is a positive sign of pregnancy. In many cases of gestation I have never failed to find it present, and in some thousands of gynecologic cases I have not seen it accompanying any other condition than

pregnancy. Jacquemin originally declared it to be a certain sign in women who have no hemorrhoids, and this statement has been confirmed by many obstetricians. It may be recognized as early as the fourth week, though it is often not well marked until the third month. Dependent as it is upon the pelvic congestion, it must increase in intensity *pari passu* with the advancing pregnancy. Toward the close of gestation the mucous surface of the vulva may be almost black in color. In the earlier weeks the discoloration may be first noticed beneath the urethral orifice, digital compression showing the sluggishness of the circulation. It is claimed that certain neoplasms may produce such a discoloration, but this must be quite exceptional. The absence of the discoloration cannot be construed as an argument against the existence of pregnancy; its presence is practically diagnostic.

The **vaginal pulse** is a frequent sign, and valuable as a presumptive symptom of early gestation. It consists in a distinct pulsation of the vaginal arteries consequent upon the high arterial tension of the pelvis. It is not invariably present, and may accompany other conditions, as fibroid tumors, extra-uterine fetation, and inflammatory pelvic conditions. Flattening of the anterior vaginal vault may likewise be noted, and was regarded by Barnes as strongly suggestive of pregnancy. It results from backward traction by the upward-tilted cervix.

Leukorrhea is of no special value as a means of diagnosis, even though the woman has never suffered from it prior to the supposed gestation. It is always present to a certain degree, but may be due to many other conditions. It is associated with more or less puffiness of the vaginal walls and vulvar tissues, and is directly dependent upon the increased pelvic congestion.

Softening of the cervix (Goodell's sign) is a very suggestive symptom, especially in primiparous women. It is due to edema of the cervical tissues beginning around the os uteri, and is present as early as the second or the third week of pregnancy. As formulated by Goodell, the rule of practice is as follows: If the cervix be as hard as the tip of the nose, pregnancy presumably does not exist, but if it be as soft as the lips, the existence of gestation is probable. It is accompanied by a considerable degree of dilatability of the cervix, especially in multiparous women. The same softening may be noted in certain pelvic inflammatory conditions, at the menstrual epochs, and accompanying the growth of soft myomata. After the fifth month the cervix will also be found to have shortened materially, and the external os points more toward the sacral hollow. By term the cervix has become fully obliterated, the internal os, however, remaining closed until labor begins.

Hegar's sign, or softening and compressibility of the lower uterine segment, is regarded by many as almost diagnostic of early pregnancy. Soft uterine myomata, however, may occasionally produce such softening of this lower segment as closely to simulate the gestation sign. Bimanual palpation is necessary to elicit the change, the right hand resting upon the abdomen just above the symphysis, while the

thumb of the left hand enters the anterior vaginal fornix and the index-finger passes far up the rectum (Fig. 2); approximation of the thumb and finger below while the uterus is depressed by the external hand will reveal the undue softening of the uterine tissues. Between the second and fifth months of gestation Hegar's sign may be regarded as one of great value. Anesthesia may be required in order to detect it in some cases, and in a large number of cases it cannot be elicited.

In **vaginal ballottement,** or the balancing of the fetus between the fingers, we have an absolutely diagnostic sign of pregnancy, which, however, is available only from the middle of the fourth to the eighth month. It is elicited by allowing the woman to stand, or by placing her upon her back with the abdominal muscles partially relaxed and the shoulders elevated. The index- and middle fingers of the left hand are introduced into the anterior vaginal fornix, while the fundus is steadied by the right hand placed on the abdominal surface (Fig. 3). The fingers in the vagina then give a sudden impulse to the anterior uterine

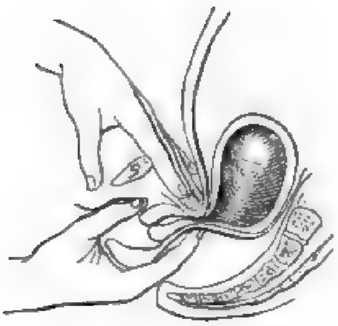


FIG. 2.—Method of eliciting Hegar's sign of pregnancy (Sonntag).

FIG. 3.—Vaginal ballottement.

wall, when the fetus, which is displaced upward in the liquor amnii, impinges upon the abdominal hand and gently falls again upon the vaginal fingers. A double thumping is thus noted. There is no other condition that could possibly produce a similar sensation, although some claim that an extra-uterine polypus with a long pedicle will respond to the test. It is not always present, however, being absent in twin pregnancy, in absence or deficiency of the liquor amnii, and in placenta prævia.

Uterine fluctuation is considered by Rasch as an important sign of early pregnancy, recognizable from the second month. It is elicited by placing two fingers of the left hand in the anterior vaginal fornix and steadying the fundus by the right hand placed on the abdominal wall. Gentle tapping by the external fingers will transmit a wave to the vaginal fingers through the agency of the liquor amnii.

Varicosities in the vaginal wall and around the vulvar orifice are indicative of pelvic congestion and are usually most prominent in the

congestion of pregnancy, because of the additional mechanical factor—pressure by the gravid uterus upon the pelvic veins. In association with other signs of the advancing gestation it possesses some significance.

An absolute diagnostic symptom is the detection, by the vaginal finger, of the fetal parts.

ABDOMINAL SIGNS.

Cutaneous discoloration of the abdomen is present in the vast majority of pregnant women. The line of discoloration—*linea fusca* or *nigra*, the yellow, brown, or black line—supplants the linea alba, and may extend as far as the xiphoid cartilage. It is most marked in brunettes, and may be altogether absent in blondes. It is by no means diagnostic, however, as boys and unimpregnated girls may present the linea fusca. The linea alba also darkens at the menstrual epochs and in certain forms of pelvic disease, as ovarian cystoma and uterine myoma.

Progressive enlargement and protrusion of the abdomen is an essential sign of pregnancy, but it is patent that the same symptom must occur whatever be the nature of the intra-abdominal growth.

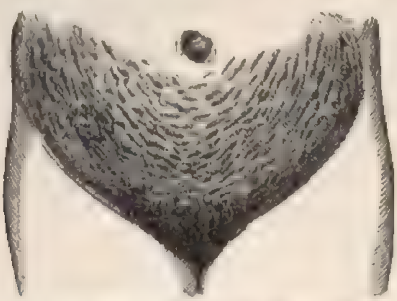


FIG. 4.—Striæ, or lineæ albicantes (Auvard).

The protrusion in pregnancy first becomes evident about the fourth month, and is most marked in women of small stature. The same errors of fallacy are to be noted as in the case of uterine enlargement.

The **striæ** are the purplish lines of discoloration that appear on the distending abdomen in the iliac region and toward the flanks (Fig. 4). They are not diagnostic of pregnancy, but may result from any excessive distention of the abdominal walls, as from cysts or ascites.

A change in the **condition of the umbilicus** is noted in pregnancy, and while this alteration may result from other causes of distention, it is most commonly associated with gestation. At the sixth month the umbilical depression is obliterated, and after that date there is a progressively increasing protrusion to term.

The **percussion-note** over the growing uterus is flat, while to the sides and above there will be found an area of tympany known as

the "coronal resonance." Any solid or fluid tumor, however, will give the same result; hence it is not characteristic.

Abdominal ballottement is an absolute sign of pregnancy, elicited by steadying the uterus with a hand placed on each side of the abdomen. An impulse given by one hand will cause the fetus to impinge upon the opposite hand; the shock is more distinct when the fetus is small and undersized.

All women are essentially **constipated,** but in pregnancy this may become an exceedingly prominent symptom. This results from the stagnation of the pelvic circulation, and is accentuated by direct mechanical pressure of the enlarged uterus upon the lower bowel. It is of value as a diagnostic means only when taken in connection with the other symptoms of early pregnancy.

Quickening and Fetal Movements.—By **quickening** is meant the first fetal movements appreciated by the mother. It usually occurs about the middle of the fifth month of gestation, but may be felt as early as the third month (twelfth week). It is peristaltic in nature, and when noticed, the woman is said to be "quick with child." All subsequent sensations of fetal life are designated as **fetal movements,** and they constitute an absolute sign of pregnancy. They are not invariably present, however; hence their absence does not indicate the absence of gestation. It is likewise possible for the woman to mistake intestinal peristalsis and choreic movements of the abdominal muscles for the sensation of quickening. The use of the stethoscope or of an anesthetic will demonstrate this condition. Active fetal movements can rarely be appreciated by the physician before the middle of the sixth month. The placing of the cold wet hand on the abdomen will sometimes cause an exaggeration of their intensity. Once detected, they steadily grow stronger with the advancing weeks. They appear either as distinct blows, as from the spasmodic movements of a fetal limb, or as a peculiar undulating movement, advancing across one side of the abdomen as the swell of a wave; this is produced by a straightening of the fetal ellipse, the back coming in contact with the uterine and abdominal walls. These movements may be absent throughout pregnancy, and the woman be delivered of a living fetus, as when there is an excess of

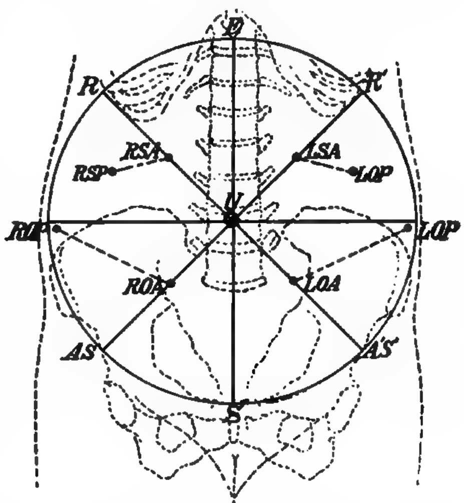


FIG. 5.—Diagram illustrating the points of maximum intensity of the fetal heart-sounds in vertex and breech presentations.

liquor amnii; or they may be suppressed for variable periods only. Several examinations should be insisted upon before the positive absence of movements is asserted. At times the movements of the fetal limbs in the liquor amnii or in contact with the uterine wall will produce faint indescribable sounds, which, if distinctly heard, are characteristic of pregnancy. It is exceptional, however, for this sound, termed the **fetal shock,** to be heard. When present, it is usually preceded by a churning sound known as the **fetal bruit.**

The **fetal heart-sounds** constitute an absolute and unmistakable sign of pregnancy. They may be detected as early as the third month, although usually not until the fifth month; from this time they grow steadily in intensity as the fetus develops. They simulate the muffled ticking of a watch placed under a pillow. The rate is about twice that of the maternal heart-beat, or from 120 to 160 beats a minute. This sign may be absent or indistinguishable in women with very fat abdominal walls, in hydramnion, when there is an excessive amount of flatus in the intestines, or when the fetal back is directed posteriorly, the sounds being most distinct over the dorsum of the fetus. Commonly they are best heard at a point below the level of the umbilicus, and to the right or left, according to the position of the fetal back (Fig. 5).

GENERAL SYMPTOMS.

Certain **urinary changes** are noted in gestation, none of which, however, is of positive diagnostic value. Frequency of micturition and vesical irritability are common, and are valuable presumptive signs of early gestation if taken in connection with menstrual suppression and moderate uterine enlargement. The presence of **kiestein,** or the formation of a fatty pellicle upon the urine which has been allowed to stand for some hours, at one time regarded as of special diagnostic value, is now known to bear no relation whatever to pregnancy.

Pressure exerted by the tumor of pregnancy upon the sacral plexus of nerves is common during the later months, and may be productive of intense neuralgic attacks or of the so-called "dead limbs." These manifestations may follow the pressure exerted by any pathologic growth.

Pigmentation of the face, forming the so-called **chloasmata, liver-blotches,** or **liver-patches,** is common in women of dark complexion. The discoloration is most marked upon the forehead and cheeks and around the eyes. If general over the face, it constitutes the *mask* of pregnancy. Such a condition may be present during menstruation and in association with uterine and ovarian disease.

Nausea and vomiting, the well-known "morning-sickness" of pregnancy, is a valuable reflex symptom, dependent upon irritation of the peripheral uterine nerves consequent upon progressive stretching of the uterine muscular fibers. It may immediately follow conception, although it usually does not appear until the sixth or seventh week. One-third of all pregnant women do not suffer from this symptom

(Giles), and, again, it may be produced by many conditions other than pregnancy. If it occur, it presents itself in 70 per cent. of the women in the first month, very rarely in the fifth and seventh months, and seldom in the second, third, or fourth month. Its duration varies from a few days to throughout the entire pregnancy, but it is rarely protracted beyond the fourth month. The nausea may be slight or severe, and generally appears immediately on rising—hence its popular name. It may occur, however, at any time during the twenty-four hours, and especially after the ingestion of food. Other conditions that may produce nausea and vomiting are uterine displacements, uterine tumors, endometritis and metritis, chlorosis, gastro-intestinal disease, ovarian and tubal disease, and menstrual retention from atresia.

Alterations of disposition and other nervous phenomena may become prominent symptoms of gestation, and are strongly suggestive because of their unwonted presence. A fretful, irritable, or moody change will be noted, and this may be associated with the morbid craving for strange and disgusting articles or for certain kinds of food. There may be noted an unusual tendency to syncope and fainting fits. Neuralgic pains are of frequent occurrence, especially Beccaria's sign—an intense pulsating pain in the occipital region. Owing to the increased amount of circulating fluid with compensatory hypertrophy of the left ventricle, the woman's pulse generally becomes somewhat accelerated, reaching not infrequently 90 or 94 beats a minute, and this may be associated with an annoying sensation of throbbing and palpitation. The cardiac symptoms are dependent upon the hydremic condition of the blood. During pregnancy the entire glandular system of the body shows increased functional activity. This is especially noticeable in the salivary glands, which may throw out an abundant watery secretion (the salivation of pregnancy).

Finally must be noted an important series of mammary changes which are strongly suggestive of the existence of pregnancy, although not absolutely diagnostic. These changes are all peculiarly well marked in primiparæ, and include general enlargement and bagginess of the organs; enlargement of the glands of Montgomery; the deposit of pigment around the nipples; the development of colostrum; erectility of the nipples; tortuosity of the superficial veins; and pricking sensations around the nipples. The pigmentation assumes the form of areolæ surrounding the nipples, and are most prominent in brunettes. The colostrum is a very valuable sign, and consists of a serolactescent fluid appearing during the third month and persisting until the third day of the puerperium. The enlargement of Montgomery's glands results in the formation of tubercles clustering around the nipples and strongly suggesting advancing gestation. The presence of milk or milky fluid is not an absolute proof of gestation, since it has been noted in the breasts of males and in young and unimpregnated women. Again, the absence of mammary changes does not contraindicate the existence of pregnancy.

THE DIAGNOSIS OF PREGNANCY.

The diagnosis of pregnancy will be made by reference to the signs just enumerated, many of which will be present in any given case. While considered separately some of these signs would not be absolutely diagnostic, when taken in combination with others a fairly presumptive diagnosis of gestation may be made. After the date of quickening the presence of the positive signs will make the diagnosis absolute. The signs to be looked for will naturally vary with the period of pregnancy to which the woman has advanced.

For convenience in examination, pregnancy is universally divided into three three-months' periods or trimesters, each of which has its own peculiar manifestations. Thus, one would not look for the positive signs prior to distention of the abdominal walls—that is, in the first trimester. In the first half of the first trimester an absolute diagnosis is not possible. There are present, however, in the first trimester, the two most important subjective signs of pregnancy: namely, menstrual suppression and nausea and vomiting. Associated with these will be found the vesical irritability, the increase in the anteroposterior diameter of the uterine body, the mammary changes, and the four soft signs: namely, the softened cervix, the soft and boggy uterine fundus, the softened lower uterine segment, and the softened and enlarged mammæ. The coexistence of these signs will be strongly presumptive of an existing gestation.

In the second trimester of pregnancy—the fourth, fifth, and sixth months—there will be developed the positive signs of the condition. Thus, by the beginning of the fourth month Jacquemin's sign (blue discoloration of the vulva and vagina) will be well marked, and Braxton Hick's intermittent uterine contractions may be detected; by the middle of the fourth month ballottement may be elicited, and quickening occurs shortly afterward; and by the beginning of the fifth month it is possible to detect the fetal heart-sounds.

In the third trimester the fetal presentation may be ascertained by vaginal exploration, and abdominal palpation will disclose the fetal outlines.

Another interesting series of correlated subjects will now arise. One of the parties in a question at law may be a young girl, presumably too young for the question of gestation to arise; or, on the other hand, she may be an elderly woman well beyond the usual date of the menopause. Thus, it may be inquired, How early is gestation possible, or what is the latest period at which pregnancy has been noted? Again, is it possible for a woman to become pregnant without a consciousness of the fact at the time of conception? Can she be pregnant any length of time and yet be unaware of her condition? Again, is there any reason why the woman should endeavor to conceal an existing pregnancy; or is there, on the other hand, anything to be gained by her by feigning to be in the pregnant state?

It is a safe general rule to consider every female from eight to eighty years of age either as pregnant or as possible of conceiving, even when other apparently impossible circumstances, aside from the question of age, may be present. Thus, the existence of an unruptured or apparently imperforate hymen will not necessarily exclude the possibility of gestation; nor will the positive affirmation of lack of penetration preclude conception. Sherwood-Dunn records the case of a woman from whom he removed both ovaries; subsequently she married and gave birth to a male child.

Precocious Pregnancy.—By this term is meant the occurrence of gestation at an unusually tender age. Probably the earliest instance of pregnancy is that famous case recorded by Tidy: A girl who had menstruated first at four years of age, conceived and was delivered of a living child when but eight years old. An allied case is recorded of a seven-pound baby being delivered from a mother but eight years and ten months old. Wharton and Stillé report an instance of a baby menstruating in her first year, and being delivered in her ninth year of a child weighing 7¾ pounds. Gleaves records the birth of a five-pound baby from a mother but ten years of age, and there are many instances recorded of girls of eleven to fourteen years giving birth to full-sized children. Wilkinson records the case of a negro girl who, when thirteen years, nine months, and five days old, gave birth to twins.

Late Pregnancy.—Cases equally as interesting as the foregoing have been filed of women conceiving late in life, and being delivered of normal children. Thus, Halles[1] records a labor occurring in a woman seventy years of age, and another in a woman of sixty-three years. Another physician records the birth of twins in a woman sixty-four years of age (Reese). It is not very uncommon to hear of women in the sixth decade giving birth to children. Hence, while the normal menopause occurs at from forty to forty-five years of age, this phenomenon may be indefinitely postponed, or ovulation may continue actively long after its associated phenomenon, menstruation, has ceased. Wolfe records an interesting case of a woman who menstruated for the first time when in her forty-third year, and continued to do so at irregular intervals until forty-five years of age, when she conceived and gave birth to a healthy child after a labor lasting but thirteen hours.

Concealed Pregnancy.—It is a much more common occurrence for a young girl to endeavor to conceal the fact that she is pregnant than it is for her to feign pregnancy for purposes of extortion or of forcing a marriage. The English law imposes no obligation to make pregnancy known. In Scotland, however, should the pregnancy be concealed and the child be dead or missing, the woman is liable to prosecution on the charge of infanticide. The concealment of birth is a much more serious misdemeanor (see Concealed Birth, p. 85).

Feigned Pregnancy.—It is rarely for purposes other than extortion or blackmail that a woman will pretend to be in gestation; hence such a claim merits the closest investigation to prevent the perpetration

[1] *Manuel complet de médecine légale.*

of an injustice. Almost invariably such women feign an advanced pregnancy—that is, after the first trimester, and, fortunately, this is at a period when the true condition of affairs can generally be readily and positively recognized. It is easier, however, to prove the absence of a gestation than it is to prove that an existing distention of the abdomen is due to pregnancy. A refusal on the part of a woman to undergo a necessary examination will be safe grounds for declaring her imposition. Occasionally hysteric women may balloon the abdomen and produce curious movements of the abdominal muscles closely simulating the fetal motions. This constitutes a form of pseudocyesis that can be diagnosed by the administration of an anesthetic.

IMPREGNATION DURING UNCONSCIOUSNESS.—UNCONSCIOUS PREGNANCY.

Here there are two closely allied and yet quite distinct propositions. The one indicates the occurrence of conception and early pregnancy in a woman, usually a married woman, without her entertaining any knowledge as to her true condition—unconscious pregnancy. The other is the occurrence in a woman, usually young and single, of gestation while the subject was in an unconscious condition, the result of deep sleep, coma, hysteria, hypnotism, or from the administration of an anesthetic. It must be recognized that intercourse during profound sleep (lethargy), either natural or the result of design, is quite possible. Such a condition will necessarily involve the question of rape. Under such circumstances it is not at all improbable for the woman to be unconscious of her delicate state during the first two or three months. After the fifth or sixth month, however, the continued menstrual suppression, together with the development of other diagnostic signs, should lead a woman with ordinary intellect to suspect her true condition. If she be a virgin, the soreness in and injuries to the vagina and vulva invariably associated with the first coitus should lead her to suspect violation immediately on a restoration to consciousness. Only in idiotic or feeble-minded women is it possible to conceive of the unconscious carrying of a child to term, although several such cases are on record.

An assumed unconsciousness is, however, quite a possible complicating factor. A woman may thus ascribe her symptoms to some pathologic growth, her labor-pains to intestinal colic, and, even when confronted by the child, deny knowledge as to the time and circumstances of its conception. Again, in rare cases of artificial impregnation of a woman by means of a syringe, conception may follow without the knowledge of the woman. When there exist motives for so pleading, the case requires the closest examination. In married women who have remained sterile for many years, or who conceive at or subsequent to the menopausal period, it is quite possible to conceive of a gestation advancing to near term without an accurate knowledge on their part of the actual state of affairs. Such women are much more prone to ascribe their condition to some pathologic affection (ovarian cystomata, ascites, uterine

fibroid) than to gestation. An examination by a qualified obstetrician will readily reveal the true condition.

Pregnancy in the Dead.—Occasionally, as for the purpose of identifying a woman or to prove her chastity, it will become necessary to make an autopsy to ascertain the condition of the genitalia. It must not be forgotten that in certain quarters there is a common practice of placing an unrelated fetus in the coffin of a woman in order to turn aside suspicion from the guilty person. The marks of gestation and delivery in the dead are to be found in the uterus, lower birth-canal, and in the ovary. An investigation of the uterine contents, even to a microscopic examination of the uterine scrapings, may become of positive value in certain cases. Owing to the excessive hypertrophy of the uterine muscles in gestation, decomposition will speedily take place in that organ after death, and it may be converted into a putrescent mass long before the other tissues yield. Quite the contrary is true of the unimpregnated uterus, and should an examination of such a body be made some months after death, the soft structures will be found well advanced in decomposition, with the exception of the uterus, which will probably be firm and resistant. This will be accepted as a positive proof of chastity. Should the woman have given birth to a child just prior to her death and the examination be made within a few days or weeks, the usual lesions in the vagina and cervix will be found, together with perineal and vulvar lacerations and contusions.

The uterine contents that would indicate a pregnancy include an ovum, embryo, fetus, or fragments thereof; decidual and chorionic débris; bones, and moles. The earlier the examination is made, the more conclusive will be the proof; but if the embryo be advanced beyond the period of ossification, its bones may be recognized even though several years have elapsed since the maternal death. Microscopic examination of the uterine scrapings will reveal decidual cells and chorionic villi, both of which are diagnostic of pregnancy.

Moles, also, if found, are proof positive of gestation. These include the placental or fleshy mole, and the uterine hydatids, or the vesicular or hydatidiform mole. The former is a thick, fleshy mass of tissue attached to the original placental site and consisting of both decidual and chorionic tissue, as shown by microscopic examination. It may include portions of an early ovum. Care must be taken not to confound such a growth with a uterine polypus, which generally consists of fibrous or muscular tissue suspended by a pedicle from some portion of the uterine wall, even as low as the cervical canal; it is not necessarily attached to the fundus, the common placental site. The vesicular mole is comparatively common, and consists of a mass of grape-like tissue filling the entire uterine canal and at places penetrating the wall of the uterus, even causing perforation. A fatty mole is merely a fragment of retained placental tissue that has undergone fatty change. Partially organized blood-clots may also be found in the uterine cavity. All the foregoing may be advanced as valuable evidence of pregnancy. Other instances, not conclusive proof of gestation, which may be found

postmortem *in utero* are true hydatids, which are exceedingly rare (not more than four or five such cases having been recorded), and the membranous formation that is shed in the rare condition known as membranous dysmenorrhea. Should any of the true products of gestation be found in the uterine cavity of a woman whose pregnancy could not have advanced beyond the third month, the indications would point to a criminal abortion, and spontaneous abortion in the first trimester is accompanied usually by the discharge of an intact ovum.

An ovary of a dead woman may show a recent scar, or *corpus luteum*, following the discharge of an ovum from a Graafian follicle. Formerly it was believed that a difference existed in the corpus luteum following an unfruitful ovulation and that associated with pregnancy. The latter was believed to be much larger in size and deeper in color. Hence arose the terms *true* and *false* corpus luteum, the former being the corpus luteum of pregnancy. It is now recognized that no diagnostic significance can be attached to the scar of ovulation. Pregnancy can occur without an appreciable corpus luteum being found, and a so-called *true* corpus luteum may accompany ovulation occurring during the growth of a uterine fibroid or any other condition, inflammatory or non-inflammatory, resulting in marked pelvic congestion, of which condition alone it is indicative, irrespective of the cause.

LEGITIMACY.—THE DETERMINATION OF SEX.—SIGNS OF DELIVERY.[1]

LEGITIMACY.

Legitimacy is defined as "the state of being born in lawful marriage."[2] The question of legitimacy from the medicolegal point of view embraces the subjects of disputed chastity, the duration of pregnancy, viability of the child, the physical incapacity of husband or wife, child-substitution, superfetation, and posthumous birth. In order to arrive at a definite conclusion in a given case medical and moral evidence, either alone or combined, may be employed. The question of illicit intercourse, with possible subsequent conception, while bearing indirectly upon the subject, is not to be included here.

It is presumed that any person born during the continuance of a lawful marriage between the mother and any man, or within a competent time after the dissolution of such marriage, is legitimate.[3] A child may, however, be shown to be illegitimate when it is possible to demonstrate that the man claimed to be the husband is practically incapable of being the father, as—(1) when he is under the age of puberty;[4] (2) when he exhibits some physical incapacity, as extreme age, or some natural infirmity, as azoöspermism; (3) when he was out of the country at the time the child was begotten, or a considerable length of time had elapsed since coitus had occurred, or from absence from home or death of the husband;[5] (4) where the impossibility is based on the laws of nature—that is, a white child is born of black parents, or vice versâ.[6] The date of the birth is the time that receives special consideration, since it is a fixed time; the date of conception cannot accurately be determined, even when there has occurred but a single intercourse the date of which is known; conception in such a case may not follow for many days, during which time the spermatozoids retain their vitality in the female generative tract. Again, the child will be pronounced illegitimate if adultery on the part of the woman can be proved and the offspring is repudiated by the husband.

[1] Grateful acknowledgment is due John C. Hinckley, Esq., of Philadelphia, for information on all legal questions touched upon in this chapter.

[2] Rawle's *Bouvier's Law Dictionary*, p. 183.

[3] Greenleaf-Lewis's edition, Philadelphia, 1896, sections 28 and 150; Chase's *Stephen on Evidence*, p. 255, second American edition, 1898.

[4] King *vs.* Luffe, 8 East 207, 1807.

[5] *American and English Encyclopedia of Law*, first edition, p. 225.

[6] Whisterlos's case in Cross *vs.* Cross, Paige Ch. New York, 139.

The evidence of illegitimacy must be clear and decided in order to disturb the presumption of legitimacy.[1] Children born after marriage, no matter how soon, are presumed to be legitimate.[2] Even if the woman be pregnant by another man and be so far advanced at the time of her marriage as to have her condition recognizable by her husband, the law takes this as a recognition on his part of paternity, and, therefore, of legitimacy (Reese). Taylor recognizes the possibility of a child being conceived prior to marriage and born subsequent to the paternal or maternal death (postmortem Cesarean section), and yet being legitimate. By the Common Law all children born out of lawful wedlock are bastards.[3] This is still the law of England. The Civil Law, however, provides that subsequent marriage of the parents renders the issue legitimate, and this rule has been enacted in many of the states of the United States—*e. g.*, Maine, Pennsylvania, Illinois, Michigan, Iowa, Minnesota, California, Oregon, Nevada, Washington, the Dakotas, Idaho, Montana, and New Mexico. In Massachusetts, Virginia, Indiana, Wisconsin, Nebraska, Maryland, Virginia, West Virginia, Kentucky, Missouri, Arkansas, Mississippi, and Arizona the father must acknowledge the child as his.[4]

Fecundity.—The question of the physical incapacity of the father is one of considerable elasticity. How young may a boy be and yet be able to fecundate? It is stated that spermatozoa generally first appear in the semen at the fifteenth or sixteenth year, and fecundity dates from this time. They may, however, be found at an earlier date. Probably the youngest case of paternity on record is that furnished by Hirst, of a boy of thirteen who impregnated his sister of fourteen. It is generally believed that sexual ability in the male ceases after the age of sixty-five. Hirst, however, mentions two authentic instances of paternity at eighty-two and over one hundred years respectively. The absence of the male or female generative organs would imply inability to propagate; in the case of a female who was so incapacitated and was presenting a child as her legitimate offspring, the question of a supposititious child (*child-substitution*) would arise.

The age of maternity is another variable factor. As we have already seen, cases of precocious pregnancy have been recorded at eight and nine years of age. In the Eastern countries maternity is not at all unusual at from ten to twelve years of age. It is rare for a woman to give birth to a child after the date of the menopause, and Barker states that women never conceive after fifty-five years of age.

Supposititious Children.—In the case of *supposititious children* it becomes the duty of the medicolegal examiner to inquire into the traces of heredity presented by the child, and the resemblance, physical or mental, to the alleged father. This will include examination as to features, voice, the gait; peculiar habits, traits, or tastes; the presence

[1] Plowes *vs.* Bossey, 31 L. J. Chan., 680, 1862.
[2] Page *vs.* Dennison, Greenleaf-Lewis's edition, Philadelphia, 1896, p. 168.
[3] *American and English Encyclopedia of Law*, second edition, p. 895.
[4] Rawle's *Bouvier's Law Dictionary*, p. 184.

of characteristic nevi or birth-marks which have appeared in successive generations of the supposed father's family. An important point for close investigation is the age of the child and the questions as to whether or not its age will coincide with the alleged date of delivery. It is quite difficult at the proper time to secure a new-born child of the proper degree of development for the case in question.

An examination of the mother is also necessary in order to reveal the signs of delivery, which should be recent or remote, according to the alleged date of birth. The difficulty of adjusting the maternal condition to the age of the child is greatest in the more recent cases. Thus, a child of one month's postnatal development would not be accepted by a careful examiner as the child of a woman whose physical signs would indicate a delivery at a period not more remote than a week. It becomes imperative, if not from a purely obstetric point of view, at least from a medicolegal standpoint, that every woman claiming to have given birth to a child shortly prior to the arrival of her physician be subjected to a physical examination. This examination will be made ostensibly to ascertain if any of the secundines remain, or if any extensive traumatism has occurred from lack of proper supervision of the labor; in reality, it will also prevent the perpetration of the crime of child-substitution should that be contemplated. If the woman or her family resist the making of such an examination, the physician would be justified in entertaining suspicions of some underhand proceeding, such as, in the wealthier classes, an attempt to secure title to an estate, and, in the lower classes, the extortion of blackmail. In case the refusal is persisted in, it becomes the duty of the physician to explain the serious medicolegal aspect that such a refusal entails, and if, then, the examination will not be permitted, his grounds for suspicion will be strengthened materially.

The Duration of Pregnancy.—In the determination of the legitimacy of a child it is very important that a knowledge be had of the normal average duration of pregnancy, the degree to which it may be prolonged (*partus serotinus*), and the degree to which it may be shortened and yet a living and viable child be born. The duration of a given pregnancy is a question incapable of accurate determination, as must be evident from the absolute uncertainty attendant upon the date of conception. If in every instance the time of the fruitful coition, or rather of the meeting of the ovum and the spermatozoid, could be ascertained, a basis could be had from which to calculate the probable date of confinement. As this accuracy is, as a rule, impossible in any given case, an approximate idea only can be obtained, and by taking the average of a large number of such approximations, an estimate of the normal duration of pregnancy can be made. There are but two factors upon which an estimation can be based, namely, a single coitus the date of which is known, and the date of menstrual suppression.

From a study of the relationship existing between menstruation and ovulation, the two phenomena not necessarily coexisting, it is patent that an error of three weeks or more may be made in a calculation

based upon menstrual suppression. The nearest that it is possible to estimate the duration of a pregnancy upon this basis is to compute 280 days—ten lunar or nine calendar months (forty weeks)—from the date of the last menstruation. This will give a date that is about the middle of a fortnight in which the labor will occur. Still, it must be borne in mind that pregnancy may begin during a period of pathologic menstrual suppression; and, again, that menstruation may continue for variable periods after conception, so that we have additional sources of error in this computation.

Merriman has collected 150 pregnancies in which the duration is counted from the date of the last menstruation. His table is as follows:

DURATION OF PREGNANCY DATED FROM THE LAST DAY OF THE CATAMENIA.

Weeks.	Days.	Number of Cases.	Percentage.
37th	from the 225th to the 259th day	5	3.33
38th	" 260th " 266th "	16	10.67
39th	" 267th " 273d "	21	14.00
40th	" 274th " 280th "	46	30.67
41st	" 281st " 287th "	28	18.67
42d	" 288th " 294th "	18	12.00
43d	" 295th " 301st "	11	7.33
44th	" 302d " 308th "	5	3.33
Total		150	100.00

The difference between the two extremes of this table is fifty-six days, and supposing every woman to have become pregnant five days before the menses, five, at least, passed the term of nine months by ten or twelve days.

It is probable that the history of a single coitus will give a more accurate means of estimating the duration of pregnancy, although here again there exists an element of doubt. It has clearly been demonstrated that spermatozoa may retain their functional activity within the generative tract for eight or ten days or longer, and during any portions of this time might fecundate an ovum. Hence, as the precise time of the meeting of spermatozoid and ovum cannot be determined, it is again impossible to ascertain definitely the duration of the pregnancy. Various obstetricians have computed the duration from a single coitus, and have obtained an average of 272 days. Thus, Faye found it to be 270 days; Ahlfeld, 271 days; Löwenhardt, 272 days; Stadfeldt, 272 days; Hecker, 273 days; Veit, 276 days; and Duncan, 275 days. The French authors usually give 270 days as the normal duration of gestation.

Reid has collected the history of 40 cases of pregnancy in women in whom impregnation was the result of a single intercourse, the date of which was accurately known. All the cases were instances of single women who dated from one coitus, or of married females whose husbands had been absent for a considerable time before the last intercourse. His table, which follows, demonstrates the varying duration of pregnancy, and, consequently, the variable date of parturition and delivery in women, even when the date of coitus is previously accurately established.

PREGNANCY IN WOMEN CALCULATED FROM A SINGLE COITUS.

Weeks.	Days.	Number of Cases.	Percentage.
38th . . .	from the 260th to the 266th day	5	12.50
39th . . .	" 267th " 273d "	7	17.50
40th . . .	" 274th " 280th "	18	45.00
41st . . .	" 281st " 287th "	6	15.00
42d . . .	" 288th " 294th "	4	10.00
Total		40	100.00

This table shows a variation of 34 days, while 18 cases, or 45 per cent., were delivered during the fortieth week, or from the two hundred and seventy-fourth to the two hundred and eightieth day.

Montgomery collected 56 cases of pregnancy in which the date of fruitful intercourse was known, as follows:

Weeks.	Days.	Number of Cases.	Percentage.
35th . . .	from the 239th to the 245th day	1	1.79
36th . . .	" 246th " 252d "	0	0.00
37th . . .	" 253d " 259th "	2	3.58
38th . . .	" 260th " 266th "	2	3.58
39th . . .	" 267th " 273d "	10	17.84
40th . . .	" 274th " 280th "	22	39.28
41st . . .	" 281st " 287th "	9	16.70
42d . . .	" 288th " 294th "	8	14.28
43d . . .	" 295th " 301st "	2	3.58
Total		56	100.00

The variation in this table is 59 days. The shortest duration of gestation is 242 days; the longest, 301 days. The greatest number of children were born in the fortieth week.

Of the 246 cases in the preceding tables, 86, or 39.96 per cent., were delivered in the fortieth week; 43 cases, or 17.48 per cent., were delivered in the forty-first week; 38 cases, or 15.45 per cent., were delivered in the thirty-ninth week. One case was delivered on the two hundred and forty-second day (thirty-fifth week), and 5 in the forty-fourth week; the variation is 64 days.

The estimation of the duration of pregnancy from the date of *quickening* is also open to appreciable error. Thus, quickening, while generally occurring when pregnancy is half completed,—that is to say, at about the middle of the fifth month, may occur as early as the twelfth week or not until the twenty-fifth week; in a few cases there may not be noted any fetal movements throughout the entire gestation. Again, intestinal peristalsis and irregular contraction of the abdominal muscles may simulate fetal movements so closely as to mislead not only the patient herself, but her attending physician also. Moreover, few women can tell the exact day, or even within fourteen days, of the appearance of quickening. Hence this is an uncertain date from which to reckon.

There is, therefore, no absolute figure that may be stated as the positive duration of pregnancy in the human female. In some women eight calendar months is the full period to which they can carry their young, while others invariably go well beyond the average of 280 days. Thus, while the average may be as stated, normal gestation may last but

240 days, or be extended to 300 days or longer. This irregularity is but carrying out the law as observed in lower animals, in whom, even when the date of a coitus is well known, a marked variation in the duration has been noted. Reese has made a careful study of the duration of pregnancy in rabbits, cows, mares, and sheep, and noted the following points: The average duration of pregnancy in rabbits is 31 days, with a variation of 8 days. The average duration in cows is 285 days; yet in a certain proportion gestation will end as early as the thirty-eighth and in others not until the fifty-first week, giving a period of difference of 90 days, or three calendar months. In sheep the average duration of pregnancy is 151 days, with variations from 145 to 171 days, giving a period of difference of 26 days. In mares the average term of pregnancy is 300 days; it may not terminate until 360 days, or even, as Tessier has noted, not until 394 days. Willer, as quoted by Velpeau, proved, by hatching chickens in an oven, that the process takes from 18 to 25 days. A more detailed account of the experiments of Tessier, in France, and of Spencer, in England, as presented by J. Y. Simpson, is as follows:

PERIOD OF GESTATION IN COWS.

Weeks.	Days.				Number of Cows. Tessier.	Number of Cows. Spencer.	Percentage. Tessier.	Percentage. Spencer.
37th,	from the 253d	to the	259th	day	6	12	1.05	1.60
38th,	" 260th	"	266th	"	8	4	1.40	0.55
39th,	" 267th	"	273d	"	51	24	8.91	2.80
40th,	" 274th	"	280th	"	166	121	29.02	16.53
41st,	" 281st	"	287th	"	202	392	35.31	52.27
42d,	" 288th	"	294th	"	105	175	18.36	23.18
43d,	" 295th	"	301st	"	27	16	4.72	2.12
44th and upward,	" 302d	"	321st	"	7	7	1.22	0.93
Total					572	751		

Joulin[1] records the case of a cow that bore almost sixteen months, parturition being impossible; and another that bore her calf fifteen months and two days. Gronier observed a cow that bore twelve months, and Numan one that bore eleven and a half months. Joulin further remarks that it has been offered as an explanation of protracted gestations that they depend on the imperfect development of the fetus, which remains in the mother's womb until it has become fit for extra-uterine life.

PERIOD OF GESTATION IN MARES.

Of 200 mares,	3	foaled	the 311th day.
"	1	"	" 314th "
"	1	"	" 325th "
"	1	"	" 326th "
"	2	"	" 330th "
"	47	"	from the 340th to the 350th day.
"	25	"	" 350th " 360th "
"	21	"	" 360th " 377th "
"	1	"	the 394th day.

The difference between the two extremes is 83 days.

[1] *Traité complet d'Accouchements.*

Protracted Gestation (Partus Serotinus).—From the foregoing statement it must be admitted that it is possible for gestation in the human female to be carried well beyond the normal duration; a knowledge of this truth is exceedingly essential for the medicolegal expert. Indeed, as J. Y. Simpson,[1] as long ago as 1855, remarked: "The obstetricians who maintain that the period of human gestation is a fixed period and can never by any possibility exceed forty weeks (as was sworn to by five doctors in the Gardner Peerage Case, London; and by six doctors in the American Court, at Lancaster Quarter Sessions, Pennsylvania), have none of them adduced any reason why the period of pregnancy should thus be stable and invariable, while all other periodic processes in the human body, as dentition, puberty, menstruation, the date of quickening, etc., are universally known and acknowledged to be apt to vary extremely. These obstetricians have offered no reasons, so far as I know, for holding that similar variations could not take place in the duration of pregnancy. Indeed, it would be against all analogies from other actions and processes in the animal kingdom to suppose that such variations did not occur in regard to the function of gestation."

Extreme care should be taken in every instance to ascertain the truthfulness of the statements offered by the interested party, and to learn what reasons, if any, exist why a legitimate offspring is desirable. The earnest desire of a woman to save her reputation or to present an heir may influence her statements, and every reasonable element of doubt should receive careful consideration. In many countries the utmost limit to which a legitimate pregnancy may extend is decided by law. Thus, in Scotland, France, and Italy, if the pregnancy exceed 300 days, legitimacy is denied; in Germany a duration of 302 days is permitted. Unfortunately, no such legislation is recognized in the United States or in England, and the disputants are placed at the mercy of the medicolegal experts. Reese states that a pregnancy lasting 317 days has been allowed in America in a lawsuit on the charge of seduction.

Many interesting cases are recorded of exceptionally protracted gestation, and the current medical literature from time to time contributes additional authentic instances. In each case it would be well to take the physical appearance of the child, together with its weight, into consideration as important evidence of the overduration of pregnancy. This increase in the size and weight of the child is, however, not always to be noted. On the contrary, as Duncan[2] remarks, we find authors stating that in these so-called cases of protracted pregnancy the child is no larger than usual or is even smaller than ordinary. Montgomery[3] also states that "although in some of these cases of protracted gestation the child was of enormous size, it by no means follows that it should be so in all such instances; and in point of fact we find it expressly

[1] *Obstetric Memoirs and Contributions*, vol. i.
[2] *Fecundity, Fertility, and Sterility*, Edinburgh, 1866.
[3] *Signs and Symptoms of Pregnancy*, London, 1856.

mentioned in some of them that the child was smaller than usual, as happened in one of Dr. Hamilton's cases; and Fodere says that in three instances in which gestation was evidently prolonged, the children were undersized and ill-thriven; while, on the other hand, the largest children are often produced where no extension of the term could have taken place." It is an incontestible fact that some children born three or four weeks before the estimated completion of pregnancy present the evidences of weight, of size, and of development of a mature infant, while a mature child *may be* small and undersized, though this is undoubtedly exceptional. Male children at term are more likely to be larger, stronger, and better developed than female children, although the average weight of all mature babies is seven and a half pounds. Necessarily the duration of the pregnancy will be unlimited when the fetus lies outside of the uterine cavity. In these cases of extra-uterine gestation the child almost invariably dies at or shortly after term, but the product may be retained indefinitely, even for a space of forty years, as is recorded in an authentic instance.

Murray [1] records a case in which the interval between the cessation of menstruation and delivery was 330 days, but the child, which was still-born, weighed only seven and a half pounds—a very average weight. In this instance it is probable that either the pregnancy was of normal duration, gestation having been ingrafted upon pathologic suppression of the menstruation, or the child had died at the normal end of pregnancy and had been retained until the time of delivery. R. Wilson [2] records a pregnancy in which 371 days elapsed between the date of the last menstruation and the date of the labor. Subtracting a possible error of 23 days for suppression from some other cause before pregnancy began, the duration would still be 348 days, or 11 months and 14 days—2½ months beyond the usual term. Even then, according to the history of the case, quickening must have occurred at the third month—an unusually early period. Velpeau records the case of a quartipara delivered on the three hundred and tenth day; Hamilton, the case of an elderly primipara, aged thirty-seven years, who carried her child to the eleventh menstrual period. Wigodosky [3] reports the history of a tertipara whose pregnancy, based on the history of menstrual suppression, lasted eleven months. Dewees mentions four women who habitually carried their children ten calendar months. Desormeaux records the case of a woman who gave birth to a child after nine and a half months of gestation. Burns has met with a case in which gestation persisted for ten calendar months and ten days, dated from the last menstruation; and La Motte mentions the case of a woman delivered after a term of at least 297 days. Montgomery observed an authentic case in which the period of gestation covered 292 days. Resnikow [4] personally observed the case of a duipara who gave birth to a male child, considerably macerated and decomposed, eleven months

[1] *British Medical Journal*, 1889.
[2] *University Medical Magazine*, July, 1890.
[3] *Medicinsk Obzrenie*, No. 2, 1896.
[4] *Centralblatt fur Gynäkologie*, No. 24, 1894.

after the beginning of her pregnancy. Acker[1] reports a case in which, dating from a single coitus, gestation persisted for 305 days, the child not exceeding in development an infant at term. Pürkhauer[2] records a case of partus serotinus on the three hundred and sixteenth day; Taussig,[3] a case of 323 days' duration; Hames,[4] a delivery 320 days after the date of menstrual suppression in a primipara, the child weighing 9 pounds 10 ounces; Holland,[5] the case of a primipara in whom 340 days were noted between the birth of the child and the date of the last normal menstruation; in this case from the date of the last coitus to the birth was 323 days, and from the date of the menstruation following the last coitus (an abnormal period lasting only 2 days) the duration was 316 days. Armstrong[6] reports the case of a woman who, in her second pregnancy, advanced to the three hundred and third day, and in her fourth pregnancy to the three hundred and nineteenth day. Reid[7] records an authentic case in which pregnancy lasted 291 to 293 days; while Simpson reports four cases that carried their young a long time beyond the usual limit. The first bore her child 336 days from the last appearance of the menses; the second, 332 days, the patient having in the meantime passed through the perils of a shipwreck; the third, 319 days; and the fourth, 324 days, the child being born 198 days—6 months and 16 days—after the date of quickening. Tarnier[8] delivered a woman 40 days after term, the macerated fetus weighing two and a half pounds. The exact date of the child's death could not be determined. Leishman mentions a case in which labor occurred on the two hundred and ninety-fifth day; and Hedrick records the case of a primipara who was delivered on the three hundred and ninth day after intercourse. Woollett reports the case of a girl, sixteen years of age, in whom gestation lasted 316 days from the date of coitus; and R. McBride reports another case with an interval of 296 days between a single intercourse and the occurrence of labor. Issmer[9] finds that the duration of pregnancy increases with each child until the ninth, and then there is a decrease. The age of the mother is also an important factor, as every pregnancy up to the thirty-fifth year of the mother's life is four or five days longer than the previous one. The social condition plays a part, as it has been found by Pinard that of 1000 pregnancies among working women, 51 per cent. were concluded before 280 days had elapsed, whereas of 1000 women without active occupation, only 34 per cent. were delivered before 280 days. These figures show the influence of rest upon the lengthening of pregnancy. Women who have been vaginally examined are on an average confined 5.2 days sooner than those not examined. Issmer has also found that the average duration of pregnancy in 912 strong women was 278.6 days; in 288 weak ones, 276.8

[1] *American Journal of Obstetrics*, 1889.
[2] *Friedreich's Blätter für gerichtliche Medicine*, 1890.
[3] *American Journal of Obstetrics*, October, 1901.
[4] *Lancet*, May 25, 1901.
[5] *Brit. Med. Jour.*, March 15, 1902.
[6] *Lancet*, 1890.
[7] *Ibid.*, 1890, vol. ii., p. 79.
[8] *Journal des Sages-Femmes*, May 1, 1894.
[9] *American Journal of Obstetrics*, October, 1901.

days. It is probably true that in about 6 per cent. of pregnant women the duration of pregnancy is over 300 days, and von Winckel's statistics show that prolongation occurs in 11 per cent. from 302 to 322 days.

Abnormally Shortened Labor.—While we have seen that pregnancy may be lengthened abnormally, it is just as true that it may terminate many days or weeks ahead of time. Indeed, this is a much more frequent occurrence than the former anomaly, and the two points of especial value to the medicolegal expert are the determination of the degree of development of the fetus at any given period, and the determination of the period of viability of a child, or its capacity to live after birth—that is, as an independent being.

It is not at all infrequent for a woman periodically to terminate her pregnancy at the eighth month. For her, eight months' growth constitutes the development to term. Hence, should the question arise, Was this child conceived prior to marriage, no coitus having been indulged in by the couple before the consummation of the vows? an examination of the physical characteristics of the infant will probably decide as to its period of development. Especially will this be simplified if the child be still-born, so that an examination of its organs and bony structures will be possible. A most instructive point to note in this connection is the length of the fetus, which is a factor bearing a more permanent relation to the development of the child than do the size and weight. There is no relationship existing, however, between the size and degree of stoutness of the mother and the size of the offspring, a large and well-developed woman often producing a small and immature baby with considerable difficulty, and vice versâ.

Recognizing the importance of the length of the fetus in determining the degree of development in a given case, Ahlfeld has formulated a rule and table whereby fetal size may be determined pretty accurately even before birth. He finds that the long axis of the fetus lying flexed in the uterus (the fetal ellipse) is nearly half the entire length of the fetus when extended. The long axis of the fetal ellipse may be determined approximately by placing one arm of a pelvimeter in the vagina against the fetal presentation, while the remaining arm rests on the extremity of the fetal ellipse in the uterine fundus. On doubling this measurement the approximate length of the fetus at the given period of pregnancy will be obtained. In the following table are given the measurements and weights of the fetus at varying periods in the latter half of pregnancy, as determined by this method:

Time.	Axes of Fetal Ellipse.	Length of Fetus.	Weight.
At the 25th week . .	17½ cm. (6.88 in.)	35 cm. (13.77 in.)	1218 gm. (39 oz.)
" 28th " . .	20 " (7.87 ")	40 " (15.75 ")	3½ pounds.
" 30th " . .	20⅝ " (8.12 ")	41¼ " (16.25 ")	4¼ "
" 33d " . .	21¼ " (8.36 ")	42⅝ " (16.78 ")	4½ "
" 34th " . .	21⅞ " (8.61 ")	43¾ " (17.22 ")	5½ "
" 35th " . .	22¼ " (8.75 ")	44½ " (17.51 ")	6 "
" 36th " . .	23⅞ " (9.40 ")	47¾ " (18.80 ")	6¼ "
" 38th " . .	24½ " (9.65 ")	49 " (19.29 ")	6½ "
" 40th " . .	25 " (9.84 ")	50 " (19.68 ")	6¾–7¼ "

There is, of course, just as much variation in new-born infants as regards size and development as in children of older growth. A fetus carried to term may be smaller and punier than a fetus of eight months' growth; and one fetus of eight months' development may be larger than another of the same age. As a rule, however, there will be present characteristic marks that will determine pretty accurately the age of development.

Paternity.—The question of paternity may arise not only in these cases of birth of a child at some time before the normal duration of pregnancy, but also in cases of posthumous birth occurring over nine calendar months subsequent to the paternal death; in cases of supposititious children advanced as legitimate heirs to an estate; and in cases of bastardy with an attempt at blackmail. This intricate question can be solved only by the presence or absence of paternal characteristics of face, form, speech, traits, and manner; or by the presence or absence of paternal physical peculiarities or defects, although the latter may be present in a given case without any possibility of paternity—namely, as a result of the occult process known as *maternal impression*, in which a peculiarity, mental, physical, or both, is stamped upon an offspring as a result of some profound mental impression made upon the mother while carrying the child. This impression may be made merely on sight by a man other than the legitimate husband, and the defect in the child does not necessarily imply illicit relations. The equally occult question of *telegony*, or the influence of a previous sire upon the children of a subsequent one through the same woman, must also be borne in mind. Such a transmission of previous paternal traits is well recognized in horses and cattle, and isolated instances have been noted of a similar character in man.

Viability.—By the viability of a child is meant the ability manifested by the child to survive as an independent being apart from its mother. The age of viability varies in divers species of animal-kind. It is conceded generally that 180 days, or six calendar months, is the age of viability for the human infant. Undoubted cases have been reported where the fetus was born just at six months of intra-uterine development. Infants born in the fifth month or even as early as the fourth month, have survived for a short time, but such infants can never be conceived as having reached the period of viability. In instances in which infants of supposedly less than six months' duration have survived, doubts must be entertained as to the accuracy of the calculation. The only reliable evidence in such cases consists in a careful expert investigation of the tissues and organs of the child if it subsequently die, or of the physical manifestations at the time of birth. Children born at this tender age (six months), if they survive the perils of infancy, are very prone to succumb in early childhood or in early adult life to diseases that children of ordinary vitality would survive.

Live Birth.—An interesting medicolegal question naturally arises in this connection—namely, what constitutes a live birth? Upon the answer to this query will often depend the decision in important trials

for the rights of inheritance and similar legal questions. It is now generally recognized that, for a live birth to be accomplished, it is necessary that the whole body be brought into the world. There must be independent circulation; the whole body of the child must have come forth from the body of the mother, but the umbilical cord need not have been separated. Reese states that "according to the laws of the United States and England neither breathing nor crying are essential to establish a live birth; the pulsation of the child's heart, or of one of its arteries, or the slightest voluntary movement, is regarded as sufficient for this purpose. In Scotland, crying is regarded as essential; in France, respiration; and in Germany, crying, attested by unimpeachable witnesses." Live birth, therefore, is to be looked upon as quite a different thing from viability. A live birth at four or four and a half months will win a law-suit, it may be, of one kind—as, for instance, a question of *tenancy by courtesy*, where a husband acquires a life interest in the property of his wife at her death, provided a living child is born during the life of the mother,—that is, not postmortem Cesarean section,—and not manifesting the traits of a monster; but it would not win a suit requiring the delivery of a viable infant in the full sense of the term as already given. It is now very well settled that a child *en ventre sa mere*, for all practical purposes tending to its own benefit, is considered as absolutely born.[1]

The Appearance of the Fetus at the Different Months of Gestation.—From the foregoing statements it becomes evident that an exact knowledge of the characteristic features of the embryo and fetus in successive months of gestation is most important in medicolegal practice. In the following classification these features are fully presented.

Fetal Development at the Successive Months of Gestation.—From the Fourth to the Twelfth Day (the Time of Entrance into the Uterine Cavity).—The embryo exists at first merely as the disk-like embryonic spot. Soon a tube appears, the primitive neural canals opening below and supporting below a globular bag. The dorsal or abdominal plates are visible. The embryo lies within the *zona pellucida*. Size of the ovum, $\frac{2}{3}$ cm. ($\frac{1}{4}$ in.); of the embryo, $\frac{1}{4}$ cm. ($\frac{1}{10}$ in.).

From the Fourteenth to the Eighteenth Day.—The embryo appears as a semitransparent, gelatinous, flocculent mass, measuring about $2\frac{1}{3}$ lines in length, or $\frac{1}{5}$ inch.

First Month.—The amnion and umbilical vesicle are fully formed; the allantois is present, but is not united with the chorion; there is but a small amount of liquor amnii; the visceral arches are distinct; the spinal canal is closed. The curved form of the fetus is noticeable. The fetal heart may be distinguished; there are primitive traces of the liver and kidneys; the nasal pits and eyes appear, as do also the intestine and the anal and oral orifices; the extremities are rudimentary. The length of the ovum is about $\frac{3}{4}$ inch; of the fetus, 1 cm. ($\frac{1}{3}$ inch or 4 to 6 lines); its weight is 20 grains.

[1] *American and English Encyclopedia of Law*, second edition, p. 719.

Second Month.—The embryo is the size of a pigeon's egg. The amnion is distended with fluid, and is in contact with the chorion. The chorionic villi are well developed at the placental site. The umbilical vesicle is small; the umbilical cord is distinct, and the umbilical vessels visible. The visceral clefts, with the exception of the first, are closed; the head forms more than two-thirds of the embryo; the eyes, nose, mouth, and ears are distinguishable; there are primitive traces of the hands and feet, which are webbed; the vertebræ are present; the form and disposition of the brain and cord can be recognized. There is beginning formation of the external genitals, although sex is not differentiated; the Sylvian fossa is present; there is a beginning of the ossification of the lower jaw, ribs, vertebral bodies, and clavicle; the circulatory system is forming; the Wolffian bodies are present, although beginning to disappear; the kidneys and suprarenal capsules are forming. The length of the fetus is 4 cm. ($1\frac{1}{2}$ inches or 15 to 18 lines); its weight is 4 grams (60 to 62 grains).

Third Month.—The embryo has attained the size of a goose-egg; nourishment takes place by means of maternal blood; the chorionic villi are lost; the placenta is formed, but is small; the umbilical cord is spiral, and about 3 inches long; the decidua reflexa and decidua vera come in contact; the pupillary membrane is present; the eyes and mouth are closed; the teeth begin to appear; the digits become distinct (they are not webbed, and show membranous nails; the toes are still webbed); the neck is distinguishable; the ribs are formed; the genital organs are very prominent, the penis and clitoris being of equal length; the uterus appears, thus distinguishing the sex; the integument is forming; the tubercula quadrigemina, optic thalami, and corpora striata may be seen; points of ossification are present in most of the bones. The length of the fetus is 9 cm. ($3\frac{1}{2}$ inches); its weight is 30 grams (450 grains).

Fourth Month.—The placenta weighs about 3 ounces. There is a formation of Wharton's jelly in the cord; the latter is two or three times the length of the fetal body; the head is about one-fourth of the body-length; there is a formation of short silvery hair upon the scalp, and of lanugo upon the body; the skin is rosy and very delicate; the mouth is open; the external ear measures $5\frac{1}{2}$ to $7\frac{1}{2}$ mm.; there are a development of the convolutions of the brain and a formation of the muscles; the pupillary membrane is quite distinct; the intestines contain meconium; the sex is well defined; ossification begins in the lower segments of the sacrum, and in the frontal and occipital bones; the liquor amnii is relatively less in quantity, and the fetus nearly fills the uterine cavity. The umbilical cord is about 7 inches in length, and is inserted above the lower fourth of the linea alba. The length of the fetus is 16 cm. ($6\frac{1}{2}$ inches); its weight is 55 grams ($848\frac{3}{4}$ grains).

Fifth Month.—The placenta weighs 6 ounces; the umbilical cord measures 12 inches; the hair and nails are fully formed; the head, heart, kidneys, and liver are disproportionately large; the vernix caseosa appears; the face is wrinkled and senile. The external ear

measures from 8 to 12 mm. in length; the pupillary membrane is still present. The eyelids begin to open; the Sylvian fossa becomes triangular; the fissures of Rolando appears; the brain weighs 720 grains (Wenzels); ossification begins in the pubis, calcis, and ischium. The length of the fetus is 25 cm. (9⅞ inches); its weight is 273 grams (10.8 ounces). Fetal movements are perceptible.

Sixth Month.—There is a beginning deposition of fat in the subcutaneous cellular tissue; the color of the body is cinnabar red (Reese); the palms and soles are purplish in tint; there is increased growth of hair, and the appearance of eyebrows and eyelashes; the head is very soft, and the fontanels wide open; the membrana pupillaris is present; the eyelids are adherent; the umbilicus is slightly above the pubis; the testicles commence to descend toward the inguinal rings; the labia project but do not cover the clitoris; there is beginning ossification of the manubrium and pubic bones; a small quantity of meconium is found in the colon; the bladder is small, hard, and pyriform. The external ear measures from 16 to 24 mm.; the Sylvian fissure is formed, and the precentral, inferior frontal, and intraparietal cerebral sulci appear. The length of the fetus is 30 cm. (10 to 11 inches); its weight is 715 grams (23 ounces).

Seventh Month.—The skin is still wrinkled and reddish, and is covered with vernix caseosa; the lanugo begins to disappear from the face; the hair on the scalp is about ⅕ inch long; the eyelids are open; the membrana pupillaris disappears; the medulla oblongata can be distinguished; the cerebral convolutions begin to form; the ears lie close to the side of the head; the external auricle measures 26 mm.; the testicles are at or in the inguinal canal; the decidua reflexa and decidua vera have now thoroughly merged into one; the finger-nails do not quite reach the fingers' ends; meconium exists in the large intestine; ossification centers appear in the astragalus and first piece of the sternum. The length of the fetus is 35 cm. (13⅘ inches); its weight is 1213 grams (39 ounces).

Eighth Month.—The lanugo begins to disappear from the face; the skin is thicker and of a more natural color; the nails are harder, but do not project beyond the finger-tips; valvulæ conniventes are found in the small intestine; the breasts often project; the liver is still very large; the left testicle is in the scrotum; the lungs are reddish; the insertion of the funis is but slightly below the middle of the body; the external auricle measures from 26 to 28 mm.; ossification begins in the second piece of the sternum and in the lower epiphysis of the femur; the brain weighs 4860 grains (Wenzels). The length of the fetus is 40 cm. (15⅞ inches); its weight is 1617 grams (4¼ pounds).

Ninth Month.—There is a great increase in the amount of subcutaneous fat; the face loses its wrinkled and senile appearance; the lanugo begins to disappear from the body; both testicles are in the scrotum; the vulva is closed; the gray portion of the brain begins to appear; the weight of the brain is 6150 grains (Wenzels); all the diameters of the fetal head are about 1 cm. (⅜ inch) smaller than at term.

The length of the fetus is 45 cm. (17¾ inches); its weight is 1990 grams (5⅓ pounds).

The Fetus at Term.—The body of the fetus at full maturity is well rounded; the lanugo has disappeared; the face has lost its wrinkles; the skin is rosy; the nails project beyond the finger-tips; the eyelashes and eyelids are well formed; the eyes are open; the bones of the cranium are in contact; the fontanels are small; the cerebral convolutions are numerous; the cuboid bone is beginning to ossify. The osseous deposit in the inferior epiphysis of the femur measures 2 or 3 lines in diameter; meconium is present in the large intestine only, which it nearly fills; the breasts are well formed and contain secretion; the bladder contains urine; the length of the foot is 8 cm. (3.14 inches); this is regarded by many as an important proof of fetal maturity. The external auricle measures 33 to 36 mm.; the ear and nose cartilages feel hard; the cord is inserted from 8 to 10 lines below the center of the body (Moreau). The length of the fetus is 50 cm. (19⅔ inches); its average weight is 2737 grams (7⅓ pounds). Children at full term may weigh only from 4 to 6 pounds, or they may, without prolongation of pregnancy, have a weight of from 12 to 14 pounds. Such a weight, however, presupposes an overextension of pregnancy. The weight is not so constant as the length of the child.

Multiple Pregnancy.—Closely allied to the question of legitimacy is the subject of multiple pregnancy, which may manifest itself in various and perplexing ways. This will include a consideration of twin pregnancy, in which one child is delivered, but the other is not born for several weeks or months subsequently; superfetation; a double uterus; and a coexisting intra-uterine and extra-uterine fetation.

Superfetation.—By this term is meant the fertilization of an ovum when there is another ovum from a previous ovulation in utero-gestation. These children may be delivered at the same birth, the one showing signs of maturity and the other of immaturity; or else both are born at full term at an interval of from one to three months, according to the difference in the dates of conception. For a long time the possibility of such an occurrence as this was absolutely denied. A few authentic cases, however, have shown that it may exceptionally occur, but only within the first three months of gestation, before the union of the decidua reflexa and decidua vera has been completed. During this time a new ovum can be fecundated, no insurmountable barrier existing to the ascent of the spermatozoid. A more difficult point to decide is the question as to the continuance of ovulation during gestation. While it is undoubtedly true that, as a rule, this process is abolished during the progress of a uterogestation, exceptions to the rule occur here just as readily as they do in all the other functions of the body. This has been proved by the delivery of twins one black and the other white, the product of different coitions; and by the presence of combined intra-uterine and extra-uterine gestation advanced to different periods, the extra-uterine product showing the more advanced development, although subjected to greater difficulty in

the securing of proper nourishment than its fellow in utero, hence being of prior formation. The mere suppression of the menstrual function during the progress of pregnancy does not necessarily indicate the abolishment of ovulation at the same time. As we have seen, the two are not interdependent.

The theory advanced by obstetricians who do not believe in the possibility of superfetation as an explanation of cases of this description is that of the existence of a twin-gestation in which one embryo, imbibing more nutriment than the other, outgrows its fellow; thus one may appear of but seven or eight months' development and the other show full maturity; or they advance the possible existence of a double uterus (*uterus bipartitus*) in which one ovum is fertilized in either uterus at successive coitions. The unmistakable evidences, however, of double conception in lower animals prove the possibility of the same in women. Thus, Reese reports the instance of a mare covered successively by a horse and an ass, and producing at the same birth a horse and a mule. It is beyond doubt, however, that twin intra-uterine pregnancy may be associated with anomalies of development such as have been mentioned. Monstrosities may result from too intimate union of the twin embryonal parts; or through some vagary of nature one embryo may be better nourished and outgrow its fellow, which may perish at an early age and undergo mummification or calcification during the growth of its fellow. Such an embryo (*fœtus papyraceus*) may be expelled at any subsequent period of the gestation, at the time of the birth of the living child, or at some subsequent period. It is also quite possible to conceive of a twin, undersized through improper nourishment, being retained in utero for two or three months longer than its fellow, until it has arrived at a proper degree of development. This would substantiate the view of those who believe that only a mature fetus is fitted by nature for birth. The further apart the births of the children, the greater the improbability of the existence of superfetation. It is known that a woman can conceive within a fortnight after delivery (Bonnar), so that it would be possible for her to be delivered of a mature fetus on a given date, and about nine months subsequently bear another mature fetus. This, however, does not constitute superfetation.

THE DETERMINATION OF SEX.

HERMAPHRODITISM.

In certain questions touching upon "live birth" and "tenancy by courtesy" an analogous subject may arise should the fetus, which may have survived or have lived for only a few minutes after separation from its mother, present abnormalities of its genital organs, or should they be so immature as to render doubtful the sex of the child. Should the offspring be monstrous, it is not considered as capable of inheriting; and should a male child be desirous, it will become most imperative in these cases of so-called hermaphroditism to determine accurately the sex.

In the true sense of the term, hermaphroditism means the presence in one individual of the generative organs of both sexes; it is by license now applied to all individuals in whom the sexual organs are so obscure or distorted as to render doubtful the individual's sex. A true hermaphrodite is exceedingly rare, but, according to Gould and Pyle, some cases have been proved such by dissection, portions or even entire genitalia of both sexes having been found in the same body.

Two distinct types of pseudohermaphroditism occur—the male and the female. In the one there will be a cleft scrotum, an ill-developed penis, perhaps hypospadias or epispadias, rotundity of the mammæ, and feminine contour; in the other a prolonged clitoris, prolapsed ovaries, grossness of figure, and hirsute appearance. As will be noted in these individuals, the anomaly in most cases consists of a malformation of the external genitals. There exists a type of hermaphroditism in which it is impossible definitely to declare the sex, even after careful dissection of the parts; these individuals have been termed *neuter hermaphrodites.*

In determining sex the presence of ovaries or testicles is the important point to ascertain. Periodic discharges of blood would indicate a female, while an escape of semen would designate the male. The absence of one testicle (**monorchid**), either congenitally or post-operative, is no bar to procreation, nor does failure of the testicles to descend (**cryptorchid**) interfere absolutely with the procreative power of the individual. In the unfortunate subjects of pseudohermaphroditism it is often impossible to come to any positive conclusion as to their sexual nature until the time of puberty. It may be then that the distinctly feminine or masculine traits will develop and assign the individual to his or her place in life. Often, however, a truly feminine individual will at this time or subsequently assume masculine features, such as the development of a beard or the deepening of the voice. The reverse may be true of male pseudohermaphrodites.

As might be expected, the legal aspect of hermaphroditism is a complicated matter. In Rome, according to the authorities already quoted, "a hermaphrodite could be a witness to a testament—the exclusive privilege of a man—and the sex was settled by the predominance of distinctive traits. If the male aspect and traits, together with the generative organs of man, were more pronounced, then the individual could call himself a man. There is a peculiar case on record in which the question of legal male inheritance was not settled until the individual had lived as a female for fifty-one years." In later life the question of sexual development or capacity plays an important rôle in deciding suits for rape, impregnation, and allied subjects.

THE SIGNS OF DELIVERY.

In the determination of a lawsuit on the plea of legitimacy it will frequently become necessary to make a physical examination of the woman to discover the presence or absence of the signs of a delivery,

either remote or recent, according to the age of the child. This investigation is much more frequently necessary than is a medicolegal investigation to ascertain the existence of pregnancy, and naturally is required in cases of concealment of birth or infanticide. The questions arising are, Is there any reason for suspecting a pregnancy and delivery, either from the physical condition of the woman herself or from the actions of her friends? and, in the case of a feigned birth with the presentation of a supposititious child, Has this woman upon her body the marks of a delivery that will correspond in date to the age of the child? In case the question is one of criminal abortion in which the woman has perished, or it becomes necessary to prove the chastity of a woman recently deceased, an examination of the body is essential to reveal the signs of parturition, premature or at term, that must exist. Naturally, then, the subject divides itself into the two great headings—the signs of delivery in the living and in the dead.

Signs of Delivery in the Living.—The expulsion of the product of conception at any time during pregnancy before the full completion of the nine months of intra-uterine life is called by the law miscarriage or abortion. No arbitrary division, such as exists in the obstetric sense of the words, is recognized between the terms miscarriage and premature labor. Obstetrically, an abortion is the discharge of the ovum at any time during the first three months of pregnancy; miscarriage is the discharge of the fetus during the second trimester of pregnancy (fourth, fifth, and sixth months); and premature labor is the discharge of the fetus between the sixth and ninth months. This distinction does not hold in a legal sense.

The **signs** that will be present depend upon the stage to which the pregnancy has advanced and the date at which the examination is made. An early abortion, between the second and third months, may leave no signs whatever, the ovum usually escaping in its entirety without lacerating the cervical tissues, which quickly contract. Probably all that the expert would have to depend upon in such a case would be a microscopic examination of the scrapings of the uterine cavity and the existence of a slight uterine enlargement. If the microscope reveals the characteristic decidual cells, which exist only in the presence of a gestation, a positive proof would be had of the woman having recently passed through an early delivery. This examination will prove fruitless if not made within the first two or three days: by that time the parts will have returned to their normal condition.

After abortion in the third or the fourth month there would persist for a few days a patulous and congested condition of the vulvar tissues, with the presence of more or less vulvar and vaginal contusions or abrasions; the cervix uteri will also present a characteristic funnel shape (Spiegelberg), and the breasts would be congested and heavy, probably showing the presence of a small amount of serous fluid (colostrum). If a septic infection has occurred at the time of delivery, the signs would persist for a longer time, and would be associated with exudate into the uterine and peri-uterine tissues. During menstruation there

will be present a certain amount of vaginal relaxation, together with a bloody discharge which must not be confounded with that of early abortion. The characteristic odor of the decidual tissue will be of value in making a diagnosis in these cases. Herold emphasizes the general anemic appearance in early pregnancy, associated with dryness of the skin and a peculiar excitement of the pulse. Very often in early abortion there will be no constitutional symptoms upon which to base a diagnosis; this is due to the slight amount of traumatism attendant upon discharge of the small ovum.

After **delivery at term** the signs that will be present are necessarily more pronounced and longer in their duration. They will be noted in an examination of the breasts, the abdominal surface, and the genital organs. Even in these cases, however, it is important that the examination be made as early as possible after labor, for the woman's tissues promptly regain their normal condition. In most cases, after the lapse of from eight to ten days, it is difficult, if not impossible, to make a positive statement that labor has occurred. For instance, in a case of alleged child-murder, two weeks after the labor the parts will have so far regained their usual condition as to present only the slight lesions that follow an abortion; the woman could readily, in such a case, deny a recent delivery at term, and claim that her pregnancy resulted in an early abortion, thereby avoiding the penalty of infanticide. Again, a complicating factor would be the existence of a twin pregnancy or of a superfetation, in which the blighted ovum was discharged, giving the physical signs of an abortion or labor, the normal pregnancy, however, continuing to term.

The breasts after a delivery at term will show some very characteristic changes. They will be enlarged, boggy, turgid, or hard and nodulated; and, for a varying period subsequent to labor, will invariably contain serous fluid or true milk. The nipples will protrude, the superficial veins will be prominent, and the areolæ will be more or less pronounced. If the secretion of the breasts be arrested, there may be noted on the more or less relaxed and pendant organs the striæ or lineæ albicantes resulting from the subcutaneous stretching. The colostrum under the microscope will show the characteristic colostrum-corpuscles—large, round, and faintly granular, and showing nuclei under the action of acetic acid.

The abdominal walls will be relaxed, wrinkled, pendulous, and scarred with lineæ albicantes and striæ. The viscera may be readily palpated, and if the examination be made within a week of the labor, the uterus will be felt as a large hard body, reaching well above the symphysis pubis. This same condition of the walls may result, however, from ascites or a large ovarian or uterine tumor, or in emaciation occurring subsequent to obesity. It is of value, therefore, only if associated with changes in the breasts and genitalia. The general surface of the body and the face will be pale; the skin moist and clammy; the pulse soft and compressible; and the general condition one of relaxation and weakness.

The genital organs are relaxed, more or less swollen, and bathed in the lochial discharges. The vagina is smooth, relaxed, and capacious, and may show recent tears or cicatrices. The perineum may be torn, granulating, or fully cicatrized, and lacerations or evidence of laceration here are very reliable signs of delivery. The cervix is enlarged, and not so compact in structure as normally. Within twenty-four hours the internal os begins to close if all the uterine contents are extruded; the external os remains patulous for several days, and may show a transverse laceration. The uterus is enlarged, as shown by the passage of the sound and by palpation, its size varying according to the time that has elapsed since the birth of the child. The lochial discharge—which may be absent after the third day, but usually continues for from a week to two weeks or longer—may be recognized by its peculiar odor and characteristic changes, and microscopically by the decidual débris. For the first three or four days it is almost pure blood, is red in color, and is known as the *lochia rubra*; it then becomes serous in nature (*lochia serosa*), and so remains for another three or four days; finally, it is almost pure pus of a yellowish or greenish color (*lochia alba*). The labia are thickened and voluminous, and may show abrasions and contusions; the commissures are less clearly defined, and the posterior usually is ruptured; the *carunculæ myrtiformes* are present, and are most prominent in primiparæ.

Signs of Delivery at a Remote Period.—All the foregoing signs, with the exception of uterine enlargement and the presence of tears and scars, will have disappeared at the expiration of from ten days to a fortnight; hence the importance of an early investigation in cases demanding a medicolegal examination. In the cases that come under observation after this period it is important, in the first place, to ascertain how long a time has elapsed since the birth of the child. After eight or ten days it is a difficult matter to state definitely that a woman has been delivered at term; after two or three months it is legally regarded as impossible to assign the date with any degree of precision, and especially is this true if the woman be a primipara. In multiparous women there are present certain signs that indicate the previous occurrence of labor. These consist in a more or less flaccid condition of the abdominal walls, with the presence of glistening white scars upon the flanks (*lineæ albicantes*) in the vast majority of cases. Mann states that in 8 per cent. of child-bearing women these scars are absent, and it must not be forgotten that they may result from causes of abdominal distention other than pregnancy, as uterine and ovarian tumors, ascites, and after such wasting fevers as typhoid and typhus. The breasts will not be firm and protuberant, as in virgins, but softer and pendulous; the nipples also will be larger and more prominent, and there will be greater discoloration of the areolæ. The vulva will be patulous, the labia relaxed, the hymen absent, the myrtiform caruncles prominent, the commissures indistinct, the fourchet absent or presenting a line of scar-formation, and in at least 35 per cent. of the cases a laceration of the perineum, more or less extensive, will be noted. The rugæ of the

vagina are obliterated, the vaginal walls are relaxed, and if a laceration of the floor exists, there may be associated a partial displacement of the anterior wall (cystocele). The cervix uteri will be larger than in the virgin, and may show scar-tissue; the os is linear, and may admit the tip of the finger. In a certain proportion of multiparous women, especially if the children have not been carried to term, these signs will be absent, and it will be impossible to distinguish the women from nulliparæ.

Signs of Delivery in the Dead.—After death from supposed criminal abortion, or in certain cases of claim upon an estate by an infant whose mother died during or shortly after delivery, it may become necessary to institute a postmortem examination of the woman to discover, if possible, the signs of parturition, recent or remote. In each case the history during life will materially assist in arriving at a safe conclusion. Interesting instances of attempted concealment of birth have been recorded in which advantage has been taken of the occasional phenomenon known as *coffin birth*. By this term is meant the postmortem expulsion of a child between the thighs of its mother as the result of the action of the gases of decomposition. Certain women desirous of avoiding the shame of exposure of illegitimacy, or after infanticide, have placed the fetus in a similar position in the coffin of a woman recently deceased, in the hope of diverting suspicion from themselves. An examination of the body of the deceased woman would speedily disclose the perpetrated fraud.

The signs of abortion after the lapse of three or four weeks are as difficult to determine as in the living. In a recently deceased woman they are as follows: If the abortion has occurred at a very early period (first or second month), difficulty will be encountered in differentiating the condition from that which accompanies menstruation; if a few days have elapsed since the delivery, the parts will have resumed much of their usual condition. Curettage of the uterine cavity and microscopic examination of the débris will be the most reliable proof of delivery, together with a slight increase in the size of the uterus. The greatest of care must be exercised not to injure the organ when removing it, and thus further complicate the process of examination. It should be closely inspected for punctures or laceration the result of attempts at criminal abortion. Accurate measurements of the length and breadth of the cavity should be made, and the increase over the normal noted. After the third month the placental site can be distinguished as a raised and reddened area of an inch or more in diameter, and placental débris can be secured. The mere presence of a blackened and nodulated condition of the uterine mucosa does not signify criminal abortion; such a condition is present after spontaneous abortion as well. The presence of corpora lutea should be noted, although it will be remembered that no medicolegal significance can be attached thereto.

As certain drugs and poisons, known popularly as abortifacients, are frequently given for illegal purposes, the examination of the woman's body would not be complete without a close inspection of the stomach,

bowels, and kidneys for lesions resulting from these irritant remedies. Caustics and other irritants have occasionally been injected into the womb for the purpose of inducing abortion, and the vagina should be examined to discover the traces of such a substance. The mere fact that, prior to her death, the woman had manifested the symptoms of septicemia is not conclusive evidence that a criminal abortion has been performed, although evidence of a strongly circumstantial nature. Septicemia not infrequently follows an abortion occurring spontaneously.

In cases of women dying during the menstrual epoch, there will be noted more or less congestion of the pelvic viscera, some swelling or edema of the endometrium, and a general softening of the uterine substance. This is quite distinct, however, from the changes noted after abortion.

The signs present in the dead body of a woman **recently delivered at term** will, as may be expected, be very pronounced. The external genital organs will be turgid and swollen, and will present the usual abrasions, contusions, and lacerations. The vagina will be dilated, with smooth and relaxed walls. The uterus will be found large, flaccid, and hemorrhagic; it will measure from nine to twelve inches in length, and from six to eight in width; the internal os will be patulous, and the cavity will contain decidual matter and clotted blood. The placental site will be prominent and gangrenous in appearance, and will show the gaping orifices of the immense uterine sinuses; the cervix does not present the normal appearance, is very thin and greatly elongated, and often presents spots of ecchymoses which are valuable diagnostic signs.

It is well to note here the size of the uterus during the varying periods of involution. By the tenth day the intra-uterine measurement is 10.5 cm.; on the fifteenth day it is 9.9 cm.; by the third week, 8.8 cm.; by the fourth week, 8 cm.; by the fifth week, 7.5 cm.; by the sixth week, 7.1 cm.; by the eighth week, 6.1 cm.; at the tenth to the twelfth week it is 6.5 cm. Seven cm. is the normal measurement of the non-pregnant uterus, so there would appear to be a physiologic hyperinvolution present for a short time after labor.

At a **remote period** after delivery, a month or more having elapsed, it is exceedingly difficult to arrive at any satisfactory conclusion. The question then would probably resolve itself into one as to parity and nulliparity, and this could be determined only by examining the size and shape of the womb. Medical science alone cannot determine this question in the dead; the history of the patient and other factors must be taken into consideration. It is well recognized that a uterus that has borne a child never regains its virginal size and weight; its walls remain thicker, and the organ is heavier, than in the nulliparous woman. The original triangular shape of the cavity is lost to a certain extent, and a rounded outline assumed. The presence of scars or lacerations around the external os, while strongly suggestive of a previous labor, are not proof positive, as they may have resulted from some operative procedure, such as dilatation of the canal.

Feigned Delivery.—Occasionally, for nefarious purposes, such as extortion, compelling marriage, or disinheriting those who have rightful claims to an estate, or simply as a result of hysteric aberration, a woman will claim to have recently given birth to a child. An examination by a medical expert will expose the fraud without any difficulty, save in the possible instance of a woman who has recently given birth to a dead child, and who has attempted to substitute for it a living one. In such a case the measures recorded under child-substitution must be resorted to in order to unmask the deception. Very rarely, for the purpose of instituting blackmail, a woman will feign an abortion. In such cases the assumption of great prostration, together with some genital irritation, would be the features looked for. A close vaginal and mammary examination would be sufficient to expose the imposition.

Unconscious Delivery.—In association with a suit for infanticide it is quite possible for the question to arise, Can a woman unconsciously give birth to a child? Many a woman has claimed that she was unaware of the process of parturition, and that through her ignorance of the state of affairs the life of the infant was lost. It is well recognized that unconscious delivery is possible, though rare, during profound sleep, during a state of coma or syncope, in an apoplectic, eclamptic, hysteric, or asphyxiated woman, and under the influence of an anesthetic, narcotic, or intoxicating liquor. A woman suffering from paraplegia might readily give birth to a child without experiencing any painful sensation, since the uterine action is absolutely independent of volition. During ordinary sleep a primigravida is not likely to be unconscious of her pain; a multigravida may occasionally confound a labor expulsive effort with a desire to evacuate the bowels, and thus may drop her child down a privy or into a commode, with disastrous effects to the fetus. Such an accident requires a statement of all the facts from the female in question. After the accident has happened the woman is not ignorant of what has occurred, and if she claim to have no knowledge of the affair, her guilt would be assumed. Of course, delivery in a moribund woman would be accomplished either artificially or spontaneously without her conscious volition. Some women who are blessed with a roomy pelvis and strong expulsive powers accomplish their delivery with a minimum of pain, or even without any sensation of suffering, and in such cases an unconscious delivery must not be conceived as impossible. The same is true of women who have given birth to many children, whereby there has resulted marked and permanent dilatation of the lower birth-canal. If the woman claim that she was unaware of her pregnant condition, such a claim would indicate either unusually deficient cerebration or a deliberate attempt to conceal her crime, especially if she were a single woman.

Concealment of Birth—Concealed Delivery.—For obvious reasons—infanticide or the preservation of reputation after illegitimacy in the marital relation—certain women will endeavor to conceal a birth in this and other countries. The law in America varies, however, and each case must be considered and construed under the law existing

where the offense is committed. In some states, if the child be still-born, the concealment of its birth is no crime. In New York and other states the knowledge obtained while attending a woman in confinement is privileged, and testimony concerning such matters is prohibited. As stated,[1] "If any woman shall be delivered of a child, every person who shall by any secret disposition of the dead body of the said child, whether such child died before, at, or after its birth, endeavor to conceal the birth thereof, shall be guilty of a misdemeanor." The real guilt lies not so much in the concealment of the birth, as in the concealment of the body, the law requiring that the bodies be given legitimate burial. It is not necessary, from a legal point of view, that the body of the child be produced to prove a case of this kind. The proofs of the delivery are obtained by a physical examination of the mother's body. In England the death of the child must be proved in order to establish the guilt of the mother. Only in case the living child whose birth had been concealed should die before the birth was registered would the woman, according to the English statute, be open to legal action. A secret disposition of the body at once proclaims the woman guilty, and those who are associated with the deed.

[1] 24 and 25 Vict., c. 100, Sec. 60.

BIRTH AND LEGITIMACY.

WHILE the state establishes the general rules for determining whether any given birth is legitimate, yet in determining the facts and applying these rules in any given case the legal profession must often call upon the medical.

"A legitimate child," says Blackstone, "is he that is born in lawful wedlock or within a competent time afterwards. '*Pater est quem nuptiæ demonstrant*' is the rule of the civil law; with us, in England, the rule is narrowed, for the nuptials must be precedent to the birth." Here one of the presumptions is noted that makes in favor of legitimacy and the peace of families, and diminishes the possibility and necessity of litigation and expert evidence. The rules for legitimacy vary with the jurisdiction, but it may be said that, in general, the parents of an illegitimate child legitimize it by subsequent marriage after or before its birth; and also that the ancient rigor of the law against the offspring of void and voidable marriages, declaring them bastards, is now pretty generally relaxed, the child of such marriages being sometimes legitimized absolutely, sometimes under the name of the parent who has entered the marriage in good faith. The laws and precedents of adoption will often give relief in cases where legitimization is impossible.

In divorce cases, also, in which the wife is defendant, in jurisdictions where the court may pass on the legitimacy of her children, it is still often necessary to overcome a strong presumption to prove illegitimacy. In every case, even when these matters may be gone into to the greatest extent, the offspring is considered legitimate unless non-access of the husband is proved—that is, if access of the husband is possible, intercourse is presumed.

On the Continent of Europe a formal written declaration of the birth of a child must be made before the authorities, and this declaration shows whether the birth was legitimate or not.

A bastard is one born out of lawful matrimony or after a competent time following it; or in a case in which it is physically impossible that offspring should result from a given marriage. In divorce and annulment cases these questions may form issues subordinate to the main issue, and are decided by the court on the evidence offered as to possibility, capacity, access, etc. Formerly, as to all children born so long after the death of the husband that, by the usual course of nature, they could not be begotten by him, there was a right, now obsolete, by the heir presumptive, to a writ "*de ventre inspiciendo*."

As to posthumous children: If a child of a woman marrying shortly after one husband's death could be the lawful child of either of two

husbands, as far as the ordinary course of events is concerned, he could, under the common law, choose either one as his father. Marriages of this sort are forbidden by codes on the Continent of Europe; in the United States and in England the paternity in such cases is purely a matter of evidence. As noticed in other connections elsewhere, access between the spouses is presumed until disproved. "Access" is coextensive in nearly all cases with "cohabitation," even when there is a formal voluntary separation between the parties.

The proving of paternity, the proof of legitimacy, and the proof of the birth of a legitimate child, living no matter how short a time, are often matters of great moment affecting the descent of real and the distribution of personal property, and radically altering devolution of estates; according to the facts involved, estates may vest and the course of inheritance be changed. Cases where the possibility of issue is extinct as being subsequent to the menopause, while not so important since fees-tail have been abolished, still are of importance in cases of courtesy, feigned delivery, supposititious and substituted children. The whole body of legal relations between parent and child may arise or not according to proofs that a woman has given birth to a child or to a particular child.

In legal proceedings as to birth or legitimacy the expert gynecologist is called upon, therefore, for opinions—sometimes based on facts or inferences in a particular case, sometimes upon hypothetic cases predicated—as to pregnancy, its inception, duration, and termination; and also as to feigned pregnancy, as to access of the husband to the wife, viability of newly born infants, delivery, etc. He is also required to support such opinions by citing facts as to the age, condition of the organs, and the general *indicia* of the real or hypothetic cases in dispute. Thus, pregnancy is in some way an element in almost every case coming under this general title.

Disregarding, for the present, investigations in the criminal courts as to abortions and infanticide, treated elsewhere, the following classification of the general legal and medical questions above set forth may be roughly formulated:

1. Was this Living Child, Asserted or Conceded to be the Illegitimate Child of this Mother, Begotten upon this Mother by this Alleged Father?—This is the query in bastardy cases proper, and a main incident in suits based on seduction, often blackmailing in character. The child may be born or be an unborn fetus of any age. The answer must be proved affirmatively, "Yes." The object of the proceedings is to ascertain if the person accused is the father of the child, in order to prevent the latter from becoming a public charge. This is done by obliging such father to give bonds—"bastardy bonds"—for the child's support under an "order of affiliation." The subject of bastardy will be treated in a separate paragraph. Medical subjects principally involved are pregnancy and delivery, real and feigned; access; capacity.

2. Did this Husband, who is prima facie Presumed by Law to have Begotten this Child—Concededly the Child of this the Wife of the Said Husband—Actually Beget this Child on Said Wife?—This is a question that comes up as incidental to the main issue in divorce suits. The answer must

be proved in the negative, "No." The result is, unless there is some statutory relief for the innocent child, illegitimacy. Here the medical questions involved are access, pregnancy (antenuptial and postnuptial, postdivorce), physical capacity.

3. Is this Child, Alleged to have been Given Birth to by this Wife (or Woman), and also Alleged to have been Begotten on Her by this, Her Husband (or by this Man), Actually Her Child by Him?—Here we have the query in cases of substitution, of supposititious children, or disputed heirship. Substitution may also be attempted to effectuate a blackmailing scheme through an affiliation proceeding; or it may be resorted to in connection with actions to establish courtesy (see Question No. 4). The medical investigations necessary under this head are as to the symptoms and signs of pregnancy, indications of feigned pregnancy, feigned delivery, age of child, physical capacity.

4. Was this Child, a Fetus concededly Legitimately Conceived, Born Alive during the Life of its Mother?—The claim of courtesy may depend upon proof of the answer to this interrogatory, and the devolution of estates under the statutes of descent and inheritance or under some instrument may be entirely changed in consequence. Tenancy by the courtesy is an estate for life by the operation of law. It arises to the surviving husband on the decease of a wife who has been seized at any time during the coverture of an estate of inheritance, either in severalty or in common, and has a child issue by him, *born alive during the life of the mother*, which child might, by possibility, inherit the same as heir to the wife. He then holds the land during his life by the courtesy of England so called.[1] In New York divorce for the husband's fault, or deed or devise of the wife, may now defeat this estate, and similar modifications of the rule have been made in other jurisdictions.

Pregnancy, Access—Presumptions of.—The uncertainty among authorities as to the exact time after insemination at which pregnancy begins, and as to the precise manner of its causation; the extreme variations of its natural duration, and the possibility of shortening it in different degrees—all present problems of great interest to the expert.

As to the first necessary element for the legitimate pregnancy of a wife—namely, the access of the husband—inasmuch as the law has established that non-access must be proved on alleging illegitimacy of offspring, therefore, as has been shown,[2] the presumption is generally conclusive of access unless total and indisputable absence of opportunity of access appears; whence it follows that pregnancy which could be caused by such presumed access is also presumed until disproved. Thus, these two presumptions fortify each other. Even antenuptial pregnancy is presumed *prima facie* to be the result of the cohabitation of the spouses before marriage. Nay, almost the slightest opportunity for intercourse between spouses is equivalent to access conclusively presumed, so that access and intercourse are practically synonymous. Nevertheless, the case might arise in which the impossibility of access resulting in intercourse would become a medicolegal question.

Access—*i. e.*, intercourse—which might have resulted in fructifying

[1] Gerard, *Titles to Real Estate.* [2] Article on Marriage and Divorce, p. 161.

an ovum may be presumed to have occurred on the last day of an opportunity for it if such presumption could harmonize with the date of delivery; yet the court will not, if it attacks legitimacy, hear argument to support a contention that the ovum was not impregnated on the first day of access—intercourse.

Again, a child born within a competent time after access—*i. e.*, after the last day of the life of the husband, or after the last day before the spouses were divorced—is *prima facie* legitimate; and the competent time may be made, in an attack on legitimacy, to begin at an early date by proving non-access or incapacity for a certain period before death or divorce. In the case of non-access before death, the character of the disease of which the husband died is an important consideration. This *competent time* is the subject of expert evidence in this country. In France it is determined by law that a child delivered within 300 or 301 days after death or divorce is legitimate. Similar rules prevail elsewhere on the Continent. For instance, if there are access and ability between the 180th and 300th day before delivery, or delivery between 180 and 300 days after access and ability, a child thus born is held to be legitimate. Continental countries also forbid marriages of widows within a given period (in France one year) after the death of the husband. As the authorities differ extremely as to the period of gestation (Winckelman placing the proportion of cases of more than 300 days' gestation at 6.8 per cent.), and as the presumption of legitimacy raised by proof of the access of the husband within a *competent time* is hard to overcome, it can readily be imagined that great opportunities are offered to unscrupulous women to give birth to heirs not begotten by their postulated husbands. The average duration of normal pregnancy is 280 days. It ranges from 240 to 320 days.

Details as to the phenomena of impregnation are still unknown, and reliance must be placed upon an average apparent duration of gestation for a starting-point and for a presumption in cases where pregnancy figures, neglecting cases of unusually prolonged or shortened pregnancy, and relying to overcome this legal presumption by medical evidence. As insemination is not proof of gestation; as it is undiscovered just in what way fructification results from coitus, and in what manner it depends upon ovulation, menstruation, or the character and action of the spermatozoa—the law has adopted the above hypotheses, which are founded upon antecedent physiologic probabilities or, at least, possibilities.

It is therefore obvious that investigation of the signs of pregnancy at its different stages may become necessary in overcoming the legal presumptions referred to, particularly in cases where it is pretended that pregnancy existed between certain dates, and where it is being simulated at the time of investigation. Such investigation—*i. e.*, as to recent pregnancy and its character—is equally important in questions of viability already discussed. But when we come to a consideration of the signs and symptoms of real pregnancy, the evidence of simulated pregnancy, or the subject of the viability of a fetus, the legal profession must turn the matter over to the medical; and for a consideration of these points the reader is referred to the text-books on obstetrics and gynecology.

ABORTION.

By abortion is understood the expulsion of the product of conception before twenty-six weeks' gestation or viability. Premature labor refers to the birth of the fetus after it is viable, but before the normal end of pregnancy. Miscarriage is a popular term designating the emptying of the womb at any time before labor, and has no scientific meaning.

By criminal abortion, or miscarriage, we understand the bringing about of the expulsion of the ovum before it is viable, for an improper purpose. By therapeutic abortion or miscarriage is meant the premature termination of pregnancy to save the life of the mother. The attempt to destroy the impregnated ovum is held to be equally guilty with the actual accomplishment. The indications for therapeutic abortion must be carefully determined by physicians of acknowledged competence, and this procedure is never employed unless other means for preserving the life of the mother have failed. The induction of premature labor, unless for sufficient reason, is strongly to be condemned, but the law seldom has occasion to deal with this offense. So soon as pregnancy is discovered, if it is determined to put an end to it, this will be done before the mother's condition is noticeable and before the fetus is viable.

CRIMINAL ABORTION.

In estimating the frequency of criminal abortion we are at once met with the difficulty that only those cases detected or brought to trial are available for statistics. Cases in which criminal abortion is followed by no complications are seldom made public. Phillips[1] endeavored to ascertain the frequency and result of criminal abortion by questions sent to practising physicians. Seventy-five physicians estimated that throughout the country in which they practised 63.5 per cent. of abortions were criminal. The average death-rate was reported as 2 per cent. The same observers estimated that 49.5 per cent. of those women having criminal abortion remained chronically ill as a result. Comparing these figures with the results of spontaneous abortion, we find the mortality of the latter to be 1.75 per cent., while the chronic disability was 38.5 per cent. These results were obtained from physicians practising in small towns and in the country, and not from specialists in large cities. Storer, of Newport, R. I., has drawn repeated attention to the progressive decrease in population in some portions of New

[1] *Transactions Maine Medical Association*, 1896, vol. xii.

England, which he ascribes largely to criminal abortion. From the Report of the Special Committee on Criminal Abortion [1] we learn that from correspondence with 100 physicians the Committee estimated that one-third of all pregnancies throughout the country end in abortion. This is estimated at not less than 100,000 yearly. A large number of these are criminal abortions, from which the Committee estimated that 6000 women die yearly. Rentoul [2] quotes the returns for twelve months of still-birth in Great Britain as being 17,335. This did not include Scotland and Ireland. It is estimated that of legitimate pregnancies, one in twenty ends in still-birth, and of illegitimate, one in ten.

Bertillon reports that the proportion of illegitimate still-births to legitimate still-births is 193 to 100. In Denmark twice as many still-births occur in illegitimate as in legitimate pregnancies. The records of city hospitals in the United States show numerous cases of puerperal sepsis following criminal abortion, and in spite of the difficulty in obtaining accurate statistics, it is evident that criminal abortion is very common.

The perpetrators of this crime are the patients themselves, midwives, professional abortionists, and physicians. The patient herself usually learns a method of direct interference with the uterus from an abortionist, and if this results successfully, she proceeds to apply it herself when another occasion offers. At most drug-stores medicines supposed to produce abortion are on sale, and many nostrums advertised to correct female irregularities are made, bought, and used to produce abortion. Midwives often advertise private lodgings for young women in trouble. When the patient enters such an establishment she is usually supposed to come for the purpose of procuring abortion. This is done either by the midwife or by a professional abortionist summoned for the purpose. Those who make a business of procuring abortion vary greatly in education and technical skill. Some understand aseptic precautions, while others are ignorant of the rudiments of surgery. They usually have offices to which the patient goes, submitting there to some operation, so called, and returning to her home to await the result. Disreputable physicians, while not avowedly abortionists, often have a considerable practice of this sort. Under a false diagnosis, usually of some form of pelvic disease, the womb is dilated and the ovum removed by curetting, or a sound is repeatedly introduced until the contents of the uterus are expelled. Abortifacient drugs are often prescribed, and also injections which the patient may administer to herself.

The modes of performing criminal abortion are manifold. Among savages direct violence is made use of. The patient is beaten, shaken, or kicked until hemorrhage from the womb shows that the ovum is detached. The administration, by the stomach, of an active irritant has long been practised. There is no drug which is an infallible abortifacient in a healthy patient. In a recent decision the courts of England held two professional abortionists guilty of deliberate fraud because they

[1] *Transactions Michigan State Board of Health*, p. 165.
[2] *British Medical Journal*, 1895.

sold drugs advertised as abortifacients. In giving judgment the Court held that there is no drug which, by its specific action, can truthfully be termed an abortifacient.

Drugs usually sold for this purpose are supposed to act in one of two ways: First, by causing the womb to contract violently, a result which follows the administration of large doses of ergot. In the second category are substances actively irritating to the genito-urinary tract, such as savin, tansy, pennyroyal, turpentine, cantharides, and others. Any substance which profoundly irritates the mucous membrane of the gastro-intestinal tract may be followed by abortion. Thus, a pregnant woman dying of arsenical poison usually aborts if life persists sufficiently long.

A very frequent method of producing criminal abortion is by direct interference with the womb or its contents. The introduction of a sound, a knitting-needle, a catheter, a piece of wire or whalebone, a glass rod, a wooden splinter or stick, a bent hair-pin, a douche cannula, or of other objects has been observed. The forcible injection of irritating fluid into the vagina or neck of the womb is often practised. Forcible dilatation of the womb is less common, because it requires more skill. For the same reason the finger is not often the instrument of a criminal abortionist.

Methods of criminal abortion vary in different countries. Bedford[1] describes in detail the methods employed in India. Here child marriage is common and abortion is frequently practised, while the large number of widows who are not allowed to remarry afford frequent examples of criminal abortion. Violent emetics, purgatives, and irritants are employed. Locally, abortion is produced by introducing battis or sticks wrapped with cotton smeared with a paste of pounded nutmeg, capsicum seeds, or croton seeds mixed with arsenic, sulphate of copper, and carbonate of potassium. The womb is dilated by the repeated use of these substances until the ovum is expelled or the finger can remove it. We are not surprised to know that where these methods are employed many fatal results follow.

A consideration of the methods of producing criminal abortion must naturally include the incidental results of these methods. Thus Barnes[2] reports the case of a woman found dead about an hour after she had been seen in good health. She had injected a solution of acetate of lead into the uterus, producing shock which proved fatal. Although she had introduced a catheter, she had not ruptured the membranes nor wounded the endometrium. There was no history of previous ill health. She had practised abortion upon herself twelve or fifteen times previously. On two occasions she had suffered severe shock, from which she rallied slowly. Air had not been introduced into the circulation, nor had the genital canal been wounded.

A not uncommon result of attempted criminal abortion is perforation of the uterus. In the *Transactions of the Chicago Gynecological*

[1] *Edinburgh Medical Journal*, 1896, No. 42, p. 422.
[2] *Transactions Indiana State Medical Society*, 1895, p. 241.

Society, March, 1895, is described a specimen of attempted criminal abortion in which the uterus had been perforated and the fetus and placenta pushed into the peritoneal cavity. Caruso[1] describes a case in which a midwife introduced a strip of root into the cervical canal to procure dilatation. It was removed a week later at a hospital clinic. Hektoen[2] describes a case in which a rubber catheter was placed in the womb and left there several days. On endeavoring to remove it, it could not be found. In trying to secure the placenta, the womb was perforated and the intestine dragged through the vagina. At autopsy the catheter was found behind the liver in the abdominal cavity. Mann[3]

FIG. 6.—Perforation of the posterior wall of the uterus, caused by the introduction into the uterus of a probe-like instrument with the intent to produce abortion (Hofmann).

reports a case in which criminal abortion had been attempted. When the patient was brought into the hospital the intestine was found in the uterus. The patient was immediately operated upon and recovered. A similar case is reported by Alberti.[4] Resnikow[5] describes the case of a young woman who learned from a midwife to introduce a rubber sound within the uterus, leaving it for several hours until abortion occurred. Instant death followed an attempt at abortion by injecting brine and

[1] *Annal. di Ost. e Gin.*, July, 1891. [2] *American Journal of Obstetrics*, July, 1892.
[3] *Ibid.*, May, 1895. [4] *Centralblatt fur Gynakologie*, 1894, No. 39.
[5] *Ibid.*, November, 1893.

soap into the uterus in a case described by Perrin de la Touche.[1] Mittenzweig[2] reports the case of a woman who visited an abortionist for the termination of pregnancy. The operator on three different occasions introduced a long tin nozzle and injected water into the uterus. The patient perished from septicemia. It was found that the nozzle had perforated the vagina behind the cervix, and thus infected the abdominal cavity, which was filled with pus.

The liability to a fatal result after criminal abortion may be greatly increased by the administration of substances producing congestion of the pelvic organs or by local treatment bringing about the same result. Intra-uterine injections of irritant substances are especially fatal, largely through shock. Seydel[3] describes the case of a woman who had taken large quantities of cooked red wine and made local applications of mustard to bring on abortion. Failing in this, she injected wood-vinegar into the uterus. The patient died in collapse after vomiting freely and having high fever and great restlessness. On postmortem examination an ulcer was found in the wall of the womb, opening into the abdomen, and extensive disintegration of the blood with jaundice and peritonitis.

Remarkable instances are on record in which foreign bodies introduced into the uterus have disappeared within the abdominal cavity. Goenner[4] describes a case in which a rubber catheter was broken within the vagina and part of it retained. Abortion followed, with pain and tenderness on the left side. Under the use of an ice-bag, opium, and laxatives the patient recovered. About a year afterward the piece of catheter was expelled from the rectum. Wylie reported to the New York Obstetrical Society, February 16, 1892, the removal of a glass rod from the neighborhood of the left kidney. The patient had produced an abortion upon herself by means of the rod, which had slipped from her grasp into the uterus. A tumor formed in the left ovarian region, and upon abdominal section the rod was found as described. The left tube and ovary were diseased and were removed. Duncan exhibited before the Obstetrical Society of London[5] a knitting-needle 9 inches long which an unmarried girl six months pregnant had thrust through her umbilicus into the uterus to produce abortion. When the surgeon in charge of the case opened the abdomen, he found the tip of the needle projecting from the fundus of the uterus. It was withdrawn with a pair of forceps, and the puncture stitched. The patient miscarried two days later, and upon the child's buttock was seen the place where the needle had penetrated. The mother recovered.

The diagnosis of criminal abortion includes the diagnosis of recent pregnancy, plus evidence of direct interference. To ascertain the former, the genital canal must be examined to detect enlargement of the uterus and the characteristic changes in that organ and in the remainder of the genital tract. Care should be taken not to omit the examination

[1] *Gaz. hebdom. de Méd.*, 1896, No. 51.
[2] *Zeitschrift f. med. Beamte*, Berlin, 1888, Bd. i., S. 225.
[3] *Vierteljahresschrift für gerichtliche Medicin*, 1894.
[4] *Centralblatt für Gynäkologie*, January, 1894.
[5] *Transactions*, vol. xxxiv., pt. 3.

of the breasts, because the changes occurring in them in the first pregnancy are especially significant.

When recent pregnancy has been diagnosticated, it will next be necessary to diagnosticate complete or incomplete abortion. In the former the signs of early pregnancy will be present, fragments of a deciduous membrane can be obtained from the uterus, but the womb will be found empty and no portion of the ovum can be obtained. When, on the contrary, incomplete abortion is present, the signs of early pregnancy exist, and, in addition, portions of the ovum or its appendages will be found in the uterus or in the discharges from the genital canal. Among these, villi of the chorion are especially significant. If clots floating in a white basin containing cold water be agitated gently, the coloring-matter of the blood will be dissolved, and the chorion may be seen like fine white moss and readily distinguished. Its fringes are very characteristic. The amnion is less easy to recognize without careful examination, as it is very thin and transparent. If subjected to microscopic study, it is found entirely destitute of nerves and blood-vessels, and composed of delicate connective tissue and cells. The recognition of the chorion may be obscured by its cystic degeneration, but no such process is likely to mask the amnion.

The decidua has a rough surface upon one side, with loose fibers showing its attachment with the glandular tissue of the womb. The decidua of pregnancy is especially soft and filled with large ovoid cells.

The positive diagnosis of criminal abortion depends upon the detection of evidence pointing to direct interference with the genital canal. Should a patient die from an irritant poison and be found to have but just aborted, there would be a strong presumption that the abortion had been criminal and that the irritant had been taken for that purpose. In asserting that evidences are found of direct interference, it must be remembered that some congenital conditions of the cervix closely counterfeit laceration. Heintzman[1] calls attention to this point, and describes the lacerations caused by dilatation and curetting for chronic endometritis in a patient never pregnant. In women in whom no operation has ever been done, congenital fissures of the cervix have been observed which might mislead an inexperienced examiner. In women who suffer from chronic fungous degeneration of the endometrium with menorrhagia, the cervix may be somewhat softened, its mucous membrane everted and fissured, the womb slightly enlarged, giving the decided impression that a possible early pregnancy has just terminated.

The fact that the uterus has been subjected to considerable manipulation is no proof that an abortion has actually been accomplished. The pregnant womb has been curetted, painted with iodin, and variously manipulated when pregnancy was not suspected, without causing abortion.

In young girls who have never menstruated, while no doubt may exist that the patient has been exposed to conception, it may still be impossible to prove pregnancy or abortion. Thus is reported[2] the case

[1] *Wiener medicinische Wochenschrift*, 1896. [2] *Samm. Gericht. Med. Ober.*, 1891.

of a girl aged twelve years and five months, who was said to have aborted. She had been exposed to conception, and had been given liquor to induce abortion. She was taken with pain and a bloody discharge. On careful examination, abortion could not be proved, but it was thought that, as the result of criminal practice, menstruation had come on for the first time, pregnancy having never been present.

In older women criminal abortion may be alleged when the menstrual flow follows direct violence. A case is reported[1] in which a woman was struck violently upon the side of the face. She alleged that she was pregnant at the time, and that on the night following the injury she aborted with a three months' ovum which was seen by no one but herself. When the case was thoroughly examined, it was found that the patient had not been pregnant at all, but that menstruation had appeared soon after the reception of the injury.

The results of criminal abortion which are perceptible are permanent injury to the health of the patient or death. By far the most important factor in bringing about this issue is septic infection. While in modern times some abortionists are careful regarding the cleanliness of their instruments, many make no pretensions to asepsis, and, as a result, all degrees of infection follow criminal abortion. In cases not fatal, pelvic exudates and adhesions leave the uterus more or less fixed, permanently enlarged, the tubes and ovaries altered in structure, and the pelvic peritoneum more or less adherent. In fatal cases septic infection proceeds rapidly from the genital canal to the peritoneum. A fatal issue is often determined by severe hemorrhage. Those who have had extensive observation state that a patient has never in their experience perished from hemorrhage following abortion; but when a patient weakened by bleeding is also infected, she falls an easy prey to the poison. The pitiful efforts which these patients make to escape detection often bring them to the verge of death. During the great storm of the winter of 1898–'99 a young unmarried woman had a criminal abortion produced upon her. While bleeding considerably she walked over a mile through heavy snow, when all street conveyance had ceased, to seek shelter in lodgings. Her condition became so desperate that those about her were afraid to keep her, and reported her to the police, who brought her to the Philadelphia Hospital, where she was admitted to the service of the writer. On admission she seemed to be dying, and the most persistent stimulation was necessary for the first twelve hours after admission. In addition she had septic infection, and was not convalescent until eight months afterward.

The mortality of criminal abortion will never be accurately known. Tardieu estimates it at nearly 50 per cent. in a large number of cases. In a smaller series he has seen it rise to 64 per cent. Jardien[2] reports 34 cases of criminal abortion, of which 22 died.

The lesions of septic infection following criminal abortion are those of the most extreme and complicated type. At that point upon the

[1] *Samm. Gericht. Med. Ober.*, 1891.
[2] *Étude Médico-Légale sur l'Infanticide*, Paris, 1868.

uterine wall pierced by the sound of the abortionist a localized gangrene is often seen. This is well described by Winter.[1] In this case the uterus was not enlarged and seemed healthy, with the exception of a small circular area upon the posterior wall. Here the tissue had broken down, and the edges were ragged and gangrenous. Through this area infection had entered the abdominal cavity.

The late effects of sepsis may suddenly cause death after criminal abortion, as illustrated in the following case. The obstetric resident of the Maternity Department of the Jefferson Medical College Hospital was summoned to a young girl found in a lodging, having just expelled a four months' embryo. The placenta was shortly afterward delivered. The case was seen by the visiting chief, and the uterus thoroughly explored and washed out. The patient apparently did well for more than a week, when, following slight exertion, she was suddenly seized with rapid feeble pulse, symptoms of shock, and pain in the abdomen. A ruptured abscess, probably tubal, was diagnosticated, and the patient was brought at once to the hospital. She failed so rapidly that no operation could be undertaken. At autopsy the diagnosis was found correct. An abscess in the right Fallopian tube had burst, causing acute sepsis. Every circumstance surrounding the girl's first illness left no doubt of criminal abortion.

In addition to the usual shock following hemorrhage after criminal abortion, death may suddenly occur following an attempt at abortion, from air-embolism. Examples of this sort are not rare in the literature of the subject. Mitchell[2] reports a case of sudden death following attempted abortion. At autopsy it was found that pregnancy of ten weeks existed. The membranes had been punctured, and the right ventricle was filled with blood mixed with bubbles of air.

A mixed infection is not uncommon after criminal abortion. Tetanus is sometimes observed in these cases. Palmer, Kuhn, Wendling, Frankel, Leverton, Bianconi, Dizel, John, Haddon, Garrigues and Simpson, Boyd, Arnold, Baumgärtel, Weemer, Kerolfi, Peacock, and Suttler (Tübingen, 1890) report cases. The local lesions show a point in the genital canal wounded by direct interference, through which the poison entered. The constitutional symptoms were those usually seen in tetanus.

The treatment of criminal abortion requires good judgment and ample experience. The conditions present when the physician first sees the case should be accurately recorded, and a careful but gentle examination be made of the genital canal to ascertain the presence or absence of lesions pointing to direct interference. If criminal abortion has been produced, each case should be considered and treated as a septic case. The uterus should be thoroughly but gently explored, and emptied and disinfected. This may be done by the finger, followed by gentle irrigation with sterile salt solution. Caution should be observed in giving these patients anesthetics, as their weakened condition may

[1] *Zeitschrift fur Geburtshulfe und Gynakologie*, 1887, Bd. xiv., S. 242.
[2] *Journal American Medical Association*, February 15, 1896.

make anesthesia dangerous. If débris be removed from the uterus, it should be thoroughly examined and its character noted. If free hemorrhage is present, the womb may be tamponed, with advantage, with sterile gauze. If the womb is so large that the finger of the physician cannot thoroughly explore it, a blunt-edged douche-curet should be gently inserted, and the wall of the womb thoroughly but gently scraped and irrigated. The constitutional treatment of the patient calls for active stimulation, purgation, the use of cold over the abdomen, abundant nourishment, and surgical nursing.

In deciding upon operation in these cases care must be taken that the indications for such interference are clear. The physician must remember that he should coöperate with justice to secure the conviction of the perpetrator. Should operation be hastily resorted to without positive indication, the defense might allege, in the interests of the perpetrator, that the physician's operation, and not the abortionist, should be held responsible for the death of the patient. Cases of criminal abortion should at once be reported to the proper authorities, who will secure all possible evidence before the patient's condition necessitates an operation from which she may not recover.

The operations necessary in the treatment of this condition are those usually employed in puerperal septic infection. Thorough curetting and disinfection of the uterus under anesthesia, opening and draining pelvic and abdominal abscesses, the removal of diseased Fallopian tubes and ovaries, and hysterectomy may be indicated. Such patients are often unfavorable subjects for operation by reason of their depressed and depleted condition following hemorrhage and nervous shock. The mortality-rate of such operations is necessarily high, and the utmost resources of surgery are often severely taxed in the effort to save the patient's life. So long, however, as the patient can take food and stimulants she may recover, and the most desperate cases sometimes result in a slow and imperfect convalescence.

Many attempts have been made to lessen the frequency of criminal abortion; in countries where the decrease of population has alarmed the government, stringent measures have been taken and every inducement offered for the legitimate increase in the population. The establishment of foundling asylums has, it is claimed, lessened this evil by caring for illegitimate offspring and thus removing the temptation for their destruction. In countries having large standing armies, where the marriage of soldiers is prohibited until a certain amount of property is possessed, illegitimate pregnancy is frequent, but the hospital cares for the mother, while the government, in the foundling asylum, assumes charge of the child. However far this may be from an ideal state of affairs, it is true that criminal abortion is not excessively frequent under these conditions. In the United States, with few foundling asylums and with many laws of Puritan origin in effect, the temptation to criminal abortion to those illegitimately pregnant must be great. It is undoubtedly true that much can be done by intelligent philanthropy to lessen criminal abortion. Cases of illegitimate pregnancy should be taken in

hand as soon as possible, and the unfortunate woman be given kind and intelligent care. She should have an opportunity to work so long as her strength permits, when she can seek confinement in a suitable maternity hospital. After her recovery, ample time should be given to her to gain strength, and the child should be kept with her. She should then either obtain employment with the child, or so arrange its care that she can see it frequently and contribute to its support. There exist in our cities societies for the aid of such cases, so that few need be without help.

A most essential reform in the prevention of abortion could be brought about by the press. There is scarcely a paper, religious or secular, which does not contain the advertisement of a means to procure abortion. In the papers of great cities the names and addresses of those who will undertake this crime are daily published and widely circulated. Few publishers have the moral courage of the late George W. Childs, who would at any time expunge from his paper an improper advertisement. Few such passed his critics, but where one had eluded their vigilance and its character became known to Mr. Childs, he instantly ordered its expulsion.

Ecclesiastic law among some sects is a distinct prevention of abortion. The Rev. Dr. Breen[1] sets forth the teaching of the church of Rome. No scientific procedure proved to be a therapeutic necessity is refused by the Church. The destruction, however, of the living embryo ecclesiastic authority unites in condemning. The writings of the Jesuits, the Edict of the Baltimore Council, and other edicts are of like nature.

Veit and other medical writers upon the subject have urged that physicians should report all cases of abortion.[2] This course is not, however, followed in many countries, and commonly only those cases are reported in which wrong-doing is suspected or in which a fatal result occurs.

This brings up the very interesting question of the responsibilities of the medical attendant in these cases. They are well set forth by Ballantyne.[3] Attention has been called to this subject in recent years by the celebrated case of Kitson *vs.* Playfair. Here action was brought for alleged slander in statements made in confidence regarding a person seen in consultation. The question of illegitimate pregnancy was raised by these statements. During the trial it was held that the physician was guilty, and that he should not have revealed the results of his professional observations. The case was not appealed.

The writer is indebted to Russell Duane, Esq., of the Philadelphia Bar, for information regarding the laws of the United States upon this point. The statements of a physician regarding his patients concerning information acquired by him in a professional capacity fall under two heads: those statements made in a court of justice and those made out of court. Regarding the former, we have in England the trial of the

[1] *Medical Record*, New York, 1895, No. 48, page 71.
[2] *Deutsche medicinische Zeitung*, 1888.
[3] *British Medical Journal*, January 22, 1898.

Duchess of Kingston.[1] Mr. Cæsar Hawkins, who had attended the Duchess of Kingston as surgeon, was asked whether he knew of a marriage between the Duchess and another. Held by Lord Mansfield that witness must answer question: "If a surgeon was voluntarily to reveal these secrets, to be sure he would be guilty of a breach of honor and of great indiscretion; but to give that information in a court of justice, which by the law of the land he is bound to do, will never be imputed to him as any indiscretion whatever."[2] In the State of Pennsylvania the following act is operative: "An Act to prevent physicians and surgeons from testifying in civil cases to communications made to them by their patients.

"SECTION I.—Be it enacted, etc., that no person authorized to practice physic or surgery shall be allowed in any civil case to disclose any information which he acquired in attending a patient in a professional capacity, and which was necessary to enable him to act in that capacity, which shall tend to blacken the character of his patient, without his consent."

Confidential communications made by a patient to his physician were not privileged at common law, but the following states have provided by statute that communications made by a patient to his physician or surgeon, and information acquired by him while attending a patient in a professional capacity, are privileged, and such physician or surgeon cannot be compelled to testify to any such facts in subsequent legal proceedings.

The following states have legislated to the above effect by the Acts mentioned: Arkansas, Stat. 1883, p. 625, Sec. 2862; California, Code C. P., 1885, Sec. 1881; Colorado, G. S., 1883, p. 1062, Sec. 4; Dakota, Comp. Laws, 1887, p. 910, Sec. 5313; Idaho, R. S., 1887, p. 679; Indiana, R. S., 1887, p. 679, Sec. 497; Iowa, Rev. Code, 1884, p. 860; Kansas, Comp. Laws, 1885, p. 645, Sec. 323; Michigan, G. S., 1882, p. 1889, Sec. 7516, Sec. 86; Minnesota, Gen. Stat., 1881, p. 792, ch. 73, tit. 1, Sec. 10; Missouri, R. S., 1879, p. 690, Sec. 4017; Montana, Comp. Stat., 1887, p. 230, Civ. Code, Sec. 650; Nevada, Gen. Stat., 1885, p. 833, Sec. 3406, Sec. 84; New York, Code of Civ. Proc. (4 Rev. Stat. p. 164), Sec. 834; Ohio, Rev. Stat., 1884, p. 1096, Sec. 5241; Oregon, Gen. Laws, 1872, p. 251; Pennsylvania Act of 18th June, 1895, P. L. 195; Utah, Comp. Laws, 1876, p. 506; Washington, Code, 1881, p. 102, Sec. 392; Wisconsin, Rev. Stat., 1878, p. 992, Sec. 4075; Wyoming, Rev. Stat., 1887, p. 590, Sec. 2589.

Aside from the absolutely legal requirments of the case, the physician has a distinct duty to his patient and to the public. He must not violate his patient's confidence, and his effort should be not only to save her life and health, but, if possible, her character. Much can be done

[1] 20 State Trials, 355, 572, 573; trial for bigamy.

[2] For a full discussion of the celebrated Playfair case the reader is referred to an article, entitled "The Medicolegal Consideration of the Playfair Case," by Andrew J. Hirsch, Esq., of the Chicago Bar, *Medicolegal Journal*, 1896, vol. xiv., page 455.

in this way by those who see many women as patients by setting forth clearly the moral and physical dangers of criminal abortion. Too much stress cannot be laid upon the fact that the crime is equally notorious whether performed in the first weeks of pregnancy or when the ovum has become a child. The absolute refusal of the physician to entertain such a proposition has its influence for good. A physician should be able to direct unfortunate women to those who can help them, and to warn and to protect them against the practice of abortion. It is a despicable act to take from these poor creatures their little savings under pretense of admitting them to some hospital for a fee; and yet we regret to say that on several occasions unmarried women have come to hospitals, having paid a fee to some physician under the belief that he had secured their admission. It is the duty of each physician to coöperate with reputable charitable institutions to secure care for these patients. Our large cities contain numerous private lying-in hospitals and midwives' houses whose sole purpose it is to deprive the patient of her money, and, if possible, her offspring of its life.

When, however, a physician is summoned to a case of criminal abortion, he has a duty to fulfil to the community as well as to the patient. Each coroner and city official may interpret his duty differently. In the main, however, it may be stated that without revealing the patient's name, he may with propriety acquaint the proper authority with the existence of the case and ask their counsel. It is often the case that only the patient knows the name of the perpetrator, and it will usually be found that she will not reveal his identity. If she recovers, the secret remains hers, and the fact of the abortion is known to her physician. If she dies or is in danger of death, upon his information the authorities will intervene.

Reputable physicians may be made the subjects of blackmail and falsely accused of committing abortion. This but emphasizes the rule which careful physicians follow, namely, to make no examination of the pelvic and abdominal organs of unknown women without the presence of a third and known person.

A designing woman may induce a physician to commit abortion by describing symptoms pointing to some disease of the womb which requires an instrumental examination. In former times, when the use of the uterine sound was common, a physician might introduce this instrument to measure the length of the womb and thus rupture the sac of the ovum, producing abortion. At present, among those best qualified, the uterine sound is rarely employed, and the interior of the uterus is never invaded without stringent necessity and every precaution. The possibility of pregnancy must never be lost sight of in conducting any pelvic or abdominal examination, and if this be kept in mind, accidental or unwitting abortion will rarely be produced by physicians.

INFANTICIDE.

By infanticide is understood the unlawful destruction of the new-born child. The question at once arises as to whether the destruction of the child during the process of birth is infanticide. Is the product of conception at this time still a portion of the mother's body, or is it a separate individual? An illustration may be found in a case described by Blöttner.[1] A physician was called to attend a case of confinement, and found the fetus in transverse presentation. He failed to turn the child, and then endeavored to remove the child piecemeal. He was unsuccessful in this, and another physician terminated the labor. The mother died as the result of her injuries, and the first physician was subjected to arrest. In his defense it was urged that he had not been guilty of infanticide, but simply of careless and inefficient practice. As the child was not born, it was not a separate individual, but a part of the mother's body. For the prosecution, the attorney recognized the contention of the defense, but urged that as the child was partly delivered when the first physician was summoned, it could not be regarded as anything but a separate entity. The subsequent decision of the Court was in favor of the physician, and he was simply fined for negligent and unskilful practice.

The precise determination of what constitutes individual life has been a matter of repeated discussion by the courts of various countries. Ordloff[2] lays stress upon the fact that the fetus does not become an independent being until it has breathed the external air into its lungs. This is the opinion of most medicolegal authorities. It can readily be seen that important issues may turn upon this point. If the fetus be destroyed purposely before birth, it might be claimed that its destruction was accidentally due to unsuccessful efforts at delivery. If the fetus died in birth, the question as to whether it can inherit property may decide the disposition of an estate. The writer recalls the case of a mother dying from convulsions, upon whom, at the moment of death, the physician in attendance performed Cesarean section. The child did not breathe, and hence by the terms of the will the mother's fortune was not inherited by the child nor its father, but passed directly to an institution. Had the child breathed in the presence of witnesses it would have inherited, and upon its death the property would have passed in whole or in part to its father.

Strictly speaking, infanticide is the intentional destruction of the new-born child and the destruction of the fetus during birth. The latter

[1] *Klinische medicinische Wochenschrift*, 1887, p. 81.
[2] *Vierteljahresschrift für gerichtliche Medicin*, 1889.

is sometimes justifiable when labor must be quickly ended in the interests of the mother. The former can never be justifiable. An interesting question arises as to whether the destruction of a monster born living is justifiable. Cases are on record in which an attendant, afraid to allow the mother to see a monster, has killed it as soon as born. Although the circumstances must be regarded as extenuating, still the action cannot be lawful. The monster is in possession of life, and cannot rightfully be deprived of it. Fortunately, in a great majority of cases monsters are incapable of independent existence, and soon perish spontaneously.

A wide difference exists in the precise determination and definition of criminal abortion and infanticide and of the penalties which follow each. In an article on the law of infanticide, Glaister[1] reviews extensively the laws of the British Empire, and points out some discrepancies in them and practical difficulties in their enforcement. As English common law is, to a considerable extent, the basis of the law of the United States, this paper may be consulted by those interested in the subject to advantage. Its conclusions do not bear directly upon our consideration of the subject.

Especial importance attaches to the diagnosis of criminal abortion and infanticide. In the nature of the case, many difficulties surround this diagnosis. First, the secrecy which the unfortunate patient strives to maintain, and the extraordinary fact that, however much she may suffer herself, she will rarely betray the person who destroyed the product of conception. In the physical nature of the case many difficulties arise. Illegitimate births often occur without medical attendance. The mother may be alone, and, struggling in the pains of labor, may fall unconscious at the moment of birth. The child's face may lay in the blood and discharges from the womb, and respiration be prevented or much impeded. If the mother be standing at the moment of birth, the child breaking its cord may fall upon the floor and sustain serious injury. During a protracted labor, while within the body of the mother, the child may sustain injuries through pressure which may cause it to perish soon after birth. Drunken mothers sometimes smother their children while in bed with them, without deliberately intending to destroy their lives. These difficulties in diagnosis can best be appreciated by the narration of typical cases which illustrate the points under consideration. Birge[2] reports the case of a young woman who gave birth at eight months to an illegitimate child, labor being brought on by a criminal abortion. The mother was alone when the child was born, and, being unable to help herself or the child, made no effort to do so, and the child perished. She attempted to conceal its body, but failed. When the case was investigated, the principal and accessory to the abortion were secured; it was held in the case of the mother that as she had given evidence for the prosecution and had been guilty of omission only she could not be charged with infanticide. Here the difficulty in

[1] *Edinburgh Medical Journal*, 1895–'96, No. 41, p. 1.
[2] *Atlantic Medical Weekly*, 1896, vol. v., p. 161.

deciding whether the child had purposely been allowed to smother or not is evident.

It occasionally happens, with patients in institutions surrounded by attendants, that an illegitimate child may die under suspicious circumstances. The following case is an illustration: The mother of the child was a young, immoral woman, confined in a hospital. When convalescent, she was allowed in the nursery where the infants were kept, and when directed to do so by the nurses, to give her child the breast. Her milk, however, failed, and the child was fed artificially, the food being prepared under careful supervision, but not by the mother. The child was taking no medicine, was gaining in weight and doing well. Although the mother was not nursing the child, she was allowed to assist in bathing and caring for it. During the night previous to its death the night-nurses reported the child well. It slept in a crib in the nursery, the mother occupying a bed in the ward. At 7 o'clock in the morning it was seen by the head-nurse and was then doing well. It had been fed at six in the morning, its usual food, and had taken it eagerly. After the head-nurse saw the child at 7 A. M., the mother was allowed to sit with the child for an hour. At 8 A. M. one of the nurses discovered that the child was ill. Its symptoms were dilatation of the pupils, redness of the eyes, frothing at the mouth, light, pea-green discharges from the intestines with mucus, and a greenish, watery vomit containing mucus. Its stools were alkaline in reaction, the abdomen was distended with gas, and the child, in spite of prompt treatment, died six hours after it was discovered to be ill. The mother strenuously objected to an autopsy, and in the absence of any positive proof, none was obtained. It is quite true that acute gastroenteritis in a young infant may result fatally in a short time, but the occurrence was somewhat remarkable, and the precise nature of the child's illness was not satisfactorily determined. Acute gastro-enteritis was evidently present, but whether the mother could have given the child an irritant substance dissolved in water during the hour in which she sat with it is very hard to determine. Irritant substances and drugs are kept strenuously under the care of the nurses in the hospital.

In sharp contrast to cases where diagnosis is so doubtful are those where the evidence of intentional infanticide is overwhelming. Zoggl[1] reports the case of a woman, illegitimately pregnant, who denied her condition until the very last, suppressed her sufferings in labor, and allowed the child to be born in an out-house. It fell into the pit of the out-house, but as the time was winter and the contents of the pit were frozen, the mother feared lest the child might survive. She accordingly removed it and strangled it with her fingers, the marks of her nails being plainly evident on the child's neck.

The difficulty in diagnosticating between precipitate birth and infanticide has already been alluded to. Teufel[2] describes the case of an unmarried woman who was taken with cramps while upon the street,

[1] *Fried. Bl. für gerichtliche Medicin*, 1888.
[2] *Vierteljahresschrift für gerichtliche Medicin*, 1896.

and who was delivered in the standing posture, the child falling to the floor and giving a cry. The mother was near a large stone at the time, and hastily seizing the child, in picking it up struck it against the stone. The child's body was subsequently buried, but afterward found. Here infanticide could not be proved. Klein collected statistics of 183 precipitate births, in 155 of which the patient was standing at the moment of delivery; in 22 she was sitting; and in 6, kneeling. From the study of these cases he concluded that fatal injury is comparatively rare. He doubts the possibility of birth occurring while the patient stands upright. Kluseman[1] describes cases of precipitate births, among them one in which a married woman chose the upright posture during labor. Another case is described of a married woman who had several times assumed this posture, a midwife kneeling beside her to prevent injury to the child. Casper describes a case in which a patient was suddenly confined while standing, the child falling upon the snow and receiving fatal injury. He saw a patient about to enter a hospital suddenly give birth upon the threshold, the child falling to the granite floor and receiving fatal injury. Hoffmann describes similar cases, in one of which the child was suddenly expelled while the patient was being lifted from a wagon and carried into the hospital. Olshausen[2] reports similar cases. Reinhard, in a dissertation published in 1871 at Marburg, collected 23 cases. Pullman describes the case of a mother overtaken by labor while in a bath, the child being born upon the floor before she could reach her bed. These cases occurred in married women who had no reason for concealment.

There is abundant evidence to show that a woman may unexpectedly and suddenly give birth while in the upright position before she has time to assume another. In these cases the cord is either torn across, or the placenta is expelled with the child. The child strikes the floor with its presenting part. Its injuries depend upon the length and nature of the cord, the distance through which it falls, and the hardness of the floor. Plannkuch[3] and Hoffmann[4] found that a weight of 1000 grams or less is sufficient to tear the cord. The longer and thinner the cord is, the more easily is it torn. It is never torn clean across, but in parts, the amniotic covering tearing first. It is possible in the case of a torn cord for the child to bleed to death, but it is not common, because the muscular wall of the umbilical arteries usually contracts and stops the bleeding. Casper holds that the child will not die if it be still attached to the placenta and that be partly separated. The cord must be severed between the umbilicus and the placenta before bleeding will occur.

From the study of 100 cases of precipitate birth Hoffmann concludes that the fetal cranium is always injured by these occurrences. Fractures and fissures of the cranial bones are the most common injuries, usually on the side of the head, very rarely on its posterior surface. The fracture or fissure is usually a radiating one. To estab-

[1] *Vierteljahresschrift f. ger. Med.*, 1897, Bd. xii., H. 2; Bd. xx., H. 2.
[2] *Monatsschrift für Geburtskunde*, 1860, 4638.
[3] *Archiv für Gynakologie*, 1875, H. 7, S. 28.
[4] *Lehrbuch*, S. 751.

lish the facts, however, that the injuries have resulted from sudden birth, the mother must be examined and also the child, to ascertain whether this has been possible. Women having large pelves, strong uteri, and widely dilated birth-canals are those patients most apt to have this experience. The smaller the child, the greater the liability also. The examination of the child should include not only its head, but also the lungs, as evidence of respiration has been found after very extensive crushing injuries to the head. Adloff[1] describes the case of a woman who asked to be taken upon a wagon filled with sacks of potatoes and going toward her home. After she had left the wagon the driver noticed blood-stains upon some of the sacks. He reported the case, and a midwife was sent to the patient, who had discovered that she had been delivered of a child. The cord was torn off. She wrapped the child in her skirts and carried it home. She placed it beside her, and it was living. She then lost consciousness, and when she regained it the child was dead. The body of the child was examined some time after its death, and when highly decomposed. Evidences of respiration were found, and the bones of the skull showed that the child had been destroyed by blows upon the head.

It is sometimes impossible to prove definitely that death in a given case was the result of direct interference. Wolf[2] reports a case in which a dead child tightly wrapped in straw was found beside a stone wall. It was traced to a servant woman, who said that the child was hers, was still-born, and that she had placed it on the top of the wall. At autopsy there was no evidence of violence, no foreign body in the air-passages, and no assignable cause for death. It was impossible to prove that the woman had destroyed the life of the infant. A case similar to the preceding is described by Kob[3] in which the body of a child wrapped in linen cloths was found in a muff-box. No signs of violence could be detected. The mother of the child was a dissolute young woman, living with her mother, a person of bad reputation. No reasonable doubt existed that the life of the child had been purposely terminated, as it was found to have breathed. The exact method employed could not be determined, but the older woman was convicted of the crime, while the younger was acquitted as being merely guilty of negligence. Undoubtedly the previous bad character of both women strengthened the suspicion against them.

An effort is sometimes made by a mother illegitimately pregnant to destroy the child by injuring its air-passages. Schiller[4] describes the case of a woman who gave birth to an illegitimate child, and who was found by another woman with the child screaming beside her. When aid was summoned, it was found that the child was dead. The mother asserted that she had become unconscious after the birth of the child, and that she had not intentionally injured it. Autopsy disclosed that the child had died from suffocation caused by hemorrhage into the bronchi. A

[1] *Vierteljahresschrift für gerichtliche Medicin*, 1891.
[2] *Ibid.*, 1889.
[3] *Ibid.*, 1895, 9, 10.
[4] *Ibid.*, 1887.

blunt instrument or the fingers had been thrust into the child's throat. The guilt of the mother was proved in this way.

It is a matter of great difficulty to diagnosticate clearly the presence of injury to the fetus received during parturition. Such may be of two kinds—that by direct mechanical violence, and injuries brought about by the administration to the mother of poisons or of drugs acting upon the fetus. The former—namely, destruction of the fetus by mechanical means—is exceedingly rare; the latter has been observed, although not commonly. Laying aside embryotomy as a completed operation, Dölger and Maschka[1] very rarely found cases of direct mechanical interference with the child before birth, with the exception of the operation of embryotomy. Casper, from the examination of many cases in which the fetus was found in out-houses, could not in a single case assert that there was bodily evidence of the use of mechanical agents before birth. Hoffmann thinks it possible to injure the head when the membranes are ruptured. The writer recalls a case in which a physician pierced the scalp of the fetus, supposing that he was rupturing the fetal membranes. The injury was slight, however, and when seen in consultation the child was doing well and probably recovered. Tardieu could collect but 5 cases. Lafargue saw 1 case; Gallard 1. Liman reports a case in which a cut was found over the crest of the fetal ilium. Olliver and Boyer saw wounds upon the fetal vertex.

It is well known that poisons taken by the mother can be communicated to the child. Mende draws attention to the use of opium to terminate fetal life, as practised in Japan. The proposition to destroy the ectopic fetus by injecting morphin into its sac is of a similar nature. Kubassow has collected instances where chloral hydrate, chloroform, opium, and digitalis were used. Walter and Gusserow experimented upon pregnant animals, not finding in the fetal blood strychnin, morphin, veratrin, curare, and secale cornutum, which had been administered to the mother. Gusserow, in repeating these experiments upon 12 pregnant women, found that potassium iodid was detected in the fetal body about two weeks after it had been given to the mother—that is, children born two weeks after the mother had taken the potassium iodid showed the presence of the drug in the fetal urine. Clouet examined the liver of a fetus born of a mother who ate matches, and found potassium in the liver substance. The iodid seemed to be the only drug readily passed from mother to child. The question arises, Does the successful treatment of syphilis in pregnancy point to the direct passage of mercury to the fetus, or is its effect upon the fetus explained by the improved condition of the mother only? The latter is the more probable supposition.

As an extenuating circumstance in infanticide and as complicating the diagnosis of this condition we must consider the mental and nervous condition of the mother. Krafft-Ebing[2] illustrates this in the descrip-

[1] *Fried. Bll. für gerichtliche Medicin*, 1892.
[2] *Ibid.*, 1889.

tion of the case of a woman whose dead child was found about ten days after birth. The cord was wrapped tightly about the fetal neck. The child was enveloped in a woollen jacket, and the body was found in a mill-pond. The patient stated that she had been deceived and deserted, and that the child was born while she was working in the garden. She tied the cord with her handkerchief, but did not remember that the cord was about the neck, nor did she remember to have placed it there. She had no intention of killing the child. After its death she wrapped it in the jacket and placed it in the mill-pond. Her employer and others stated that her mental condition had lately been much disordered. Her mother was a cataleptic. The patient had always suffered greatly at her menstrual periods, and had recently declared that she would commit suicide. Under the circumstances, she was not convicted of wilful infanticide.

In determining the question of infanticide it must be remembered that a considerable quantity of mucus is often present in the child's throat at the time of delivery. Rancher reports[1] a case in which a still-born child was examined to determine infanticide. Death had undoubtedly resulted from suffocation. There was, however, a large amount of greenish mucus in the child's pharynx, and it was impossible to determine that the suffocation of the infant had been intentional. He also reports a case which illustrates the difficulty in proving the commission of infanticide. An infant was found immediately after birth by a midwife, and said by the mother to have been still-born. It was illegitimate. The only evidence of infanticide present was a bluish spot upon the child's throat. At autopsy not the slightest trace of violence could be detected, and the crime could not be proved.

Suffocation is usually effected by holding the hand over the child's mouth or by placing some soft material over the face. Fielitz[2] reports 3 cases of infanticide in which the perpetrators described the methods employed. In one instance the hand was held over the child's mouth; in another a feather bed was placed over the infant; and in the third, the child's head was wrapped in an apron.

A mother, in endeavoring to extract the child from her body, may cause injuries apparently intentional. Comrick[3] illustrates this point by the case of an unmarried servant woman, delivered alone of a child, which was found living, under the steps of a closet. The child was covered with blood, its mouth wide open, its tongue directed toward the right side. It died a half hour after it was found. On examination the lower jaw was found broken in several places, and sufficient hemorrhage had ensued to cause death. The patient stated that in birth she had placed her thumb in the child's mouth so soon as the head was born, and pulled violently to hasten delivery. She was convicted of criminal carelessness, but there was no evidence of infanticide.

Carbolic acid is occasionally employed to destroy the infant. Coester[4] observed a case in which a mother admitted to have given her infant a

[1] *Fried. Blt. für gerichtliche Medicin*, 1888.
[2] *Vierteljahresschrift für gerichtliche Medicin*, 1891. [3] *Ibid.*, 1896. [4] *Ibid.*

teaspoonful of clear carbolic acid solution, which had been prescribed for her to be diluted and used in vaginal douches. Cohn, in the same journal, describes a case in which a compress saturated with pure carbolic acid was placed over the child's umbilicus. Death resulted.

The result of infanticide is the destruction of the infant's life. It is impossible actually to estimate infant mortality from this cause. The reports in eleven urban districts in England from 1871 to 1875 show a mortality among illegitimate children of 388 in a thousand, while among legitimate infants in the same districts the mortality was 192 in a thousand. Other statistics lead us to believe that the illegitimate infant is four times in danger of death to one chance which the legitimate child must meet. The maternal mortality of infanticide results but indirectly in these cases. Hemorrhage and septic infection are the principal causes, with an exceptional serious result from shock. The circumstances of illegitimate birth, with the profound nervous depression, loss of blood, and anemia, frequently leave these patients in bad general health.

The question naturally arises, What can be done to diminish the prevalence of this crime? It was allowed in some countries in ancient times. Children were sometimes offered as sacrifices to heathen deities. Children were offered on the altar of Moloch, and this was done by Solomon and others. In Carthage the people wished to sacrifice Hannibal's child to their gods. The Romans checked infanticide for religious rites, but allowed it for other reasons. Parents desiring to rid themselves of their children sent them to a place appointed, where any one could go and secure a child if desired. Those that were not taken were killed. Tacitus declares that the Germans did not practise this crime, and under the Roman Republic it became less common. In the time of Trajan the state adopted and supported children which otherwise would have been killed. In the third and fourth centuries A. D. infanticide became again common in the Roman Empire. The Bishop of Rome and Constantine made infanticide punishable by death. Poor parents were assisted by the state in maintaining their offspring. Parents could dispose of their children if they so desired. These laws, however, were very difficult to enforce. During the middle ages infanticide was punished as a religious offense. New-born children without homes were laid upon the doorsteps of monasteries and clergymen's houses. As all charity was dispensed by the clergy, this was an appeal for help for the child. In 1530, in the reign of Karl V., a person convicted of infanticide was sentenced to be drowned. This was the common penalty throughout Europe. Toward the close of the eighteenth century milder punishments were decreed throughout Germany, and efforts were made through Europe to lessen the crime by helping the poor and by establishing foundling asylums. In the Prussian estates foundling establishments are not allowed because of their great expense and high mortality. Mother and child are cared for together, and the father of the child is obliged to contribute to its sup-

port. Baby-farming or the boarding-out of infants is attended with a mortality estimated at over 30 per cent.

At the present time modern philanthropy endeavors to prevent infanticide in two ways: The unfortunate mother so soon as her pregnancy is discovered is aided to obtain suitable work, medical attendance, and, if necessary, financial aid. She is warned against criminal abortion, and is kept under observation by the agent of a charitable society. Her confinement takes place in a maternity hospital, and every effort is made to encourage her love for the child. If the father of the child is a suitable person, the endeavor is made to bring about a marriage. Should this fail, legal proceedings may be instituted to compel him to aid in the support. When the mother is convalescent, she is assisted to get work in the country, taking her child with her, and is kept under observation by a proper visitor. When the child grows older it is placed in a free school. Others maintain that a foundling asylum gives the best chance for the mother and a fair chance for the child. By those who favor this plan the child is taken from the mother soon after birth, is placed in a foundling asylum, artificially fed, and should it survive, is adopted or placed in a public school. The mother returns to her work and has but little, if any, knowledge regarding the child. The father may be obliged to pay something to the mother, but he does not follow the fate of the infant. This course is open to the very grave objection of great infant mortality. Many think that the influence upon the mother of caring for her child through the first weeks of its life should not be neglected. The former seems the better plan, and is successfully carried out in many of our large cities.

Recent studies in the pathology of the fetus are of importance as bearing upon the diagnosis of the respiratory act. It has been commonly supposed that solidification of the fetal lung and the persistence of this condition after birth indicated that respiration had never been performed. Cases are on record, however, where a child has breathed after birth, where, because of injuries during birth, it has suffered from pulmonary hemorrhage and has perished without intentional violence. It must be remembered that interference with the fetal circulation during labor produces respiratory movements and considerable alterations in the blood tension of the fetus. Attention must also be called to the fact that, in attempting to resuscitate an asphyxiated child by some of the methods commonly employed, air may be forced into the lungs of an infant that never breathed spontaneously. From an examination of the infant's body alone it may be impossible to assert that a respiratory act did or did not take place. The importance of this fact upon expert testimony based upon an autopsy only must be apparent. The simple hydrostatic test of the infant's lung is in no way sufficient to establish the presence or absence of respiration, and hence a verdict of infanticide based largely upon this might be exceedingly wide of the mark.

As an illustration of the fact that the lungs of the child may be found practically solidified after respiration, we insert the postmortem findings upon the case of an ill-nourished infant, born spontaneously,

and seen to breathe repeatedly during the seven and a half hours of its life. The case occurred in the maternity ward of the Jefferson Medical College Hospital, under the observation of the House Staff, and the autopsy was made by the Pathologic Staff of the hospital. Had it been necessary, abundant witnesses could have been procured to prove the occurrence of respiration. The autopsy notes are as follows:

A white male child, aged seven and one-half hours. The subject shows postmortem lividity on the face and upper and anterior portions of the body. Postmortem rigidity scarcely perceptible.

Brain and its meninges are normal. On section, the abdominal organs were found in their normal positions. The cavity contained a normal amount of clear yellow fluid. All the abdominal organs were normal. However, a Meckel's diverticulum about 1.5 cm. long was found 25 cm. above the ileocecal valve. The pleural and pericardial cavities were normal in every particular. With the exception of the heart muscle being somewhat flabby, the organ was normal. Both lungs were in a state of complete solidification. They did not crepitate, and sank when placed in water. While not exactly resembling it, the appearance was more that of the stage of red hepatization of pneumonia than of any other condition. Cultures were made from the lung and heart on blood-serum tubes, but bacteria did not develop.

Microscopic Examination of the Lungs.—Pieces of the lungs were fixed in Heidenhain's mercuric chloride solution and embedded in paraffin. Sections were stained in hematoxylin and with eosin and acid orcein, and by the methods of Van Gieson and Weigert.

On examination, the pleural covering of the lung is found uniformly considerably thickened, as are also the trabeculæ sent into the lung substance from the pleura. This thickening is due to interstitial hemorrhage; the blood, which contains no more than the normal number of leukocytes, seems to have penetrated between every fiber of the elastic and collagenous tissues which make up these structures, and, in most cases displacing the epithelial cells on the outer surface of the pleura, has gained the free surface of the membrane. The walls of the blood-vessels of the trabeculæ have been included in this hemorrhagic process; the inner coats of these vessels, however, do not seem to have suffered as much as the surrounding tissues. The elastic and collagenous fibers stain faintly; it seems probable that this is the result of hemorrhage. The connective-tissue cells exhibit no change.

In every particular the alveolar walls present the changes which have been noted as occurring in the pleura and trabeculæ. The tissues are so crowded with blood that it is impossible to make out the capillary walls, but nevertheless it cannot be doubted that the abundant and uniform hemorrhage must have been caused by rupture of these vessels. The epithelial lining of the air-sacs is entirely shed off. The lumina of the air-vesicles is considerably decreased by the swelling of their walls. For the reason that the size of a section of an air-vesicle will depend upon the point where it is cut, it is impossible to say with certainty just how much smaller they are in this instance. The lumina of the vesicles vary from 40 to 130 mm., with a general average of about 80 mm. in diameter. Sections from the lung of another infant show the average size of the vesicles to be about 110 mm.; the smallest of these are 70 mm. in diameter. The air-sacs are partially empty, but practically all contain numerous red and white cells, and here and there swollen epithelial cells. The last vary in size from 10 to 24 mm., and are rounded or oval in form. One or more vesicular nuclei are generally present, but some of the cells contain none; the protoplasm of these cells is intensely acidophilic. In many cases it seems to have taken the place of the epithelial cells on the walls of the air-vesicles, and is possibly the degenerate remains of these cells. If this is the case, the epithelial cells must have been in a great degree broken down before the birth of the infant. No fibrin was found in any of the sections. The epithelial cells lining the bronchi are in almost all cases detached from their basement membranes, but they appear otherwise normal. The smaller bronchi are usually filled with blood. No bacteria were present.

The clinical history of the mother and of the labor is as follows:

The mother of the infant was a primipara, aged nineteen. She was unmarried, was in an institution for the care of unfortunate women, and was taken in labor without the knowledge of the matron. When her condition was discovered, labor was well advanced. She was brought by ambulance to the maternity department of the hospital, where it was found that the child's head was on the pelvic floor. In a few moments the child was easily expelled. It was a male weighing four pounds three-quarters of an ounce, showed no apparent abnormalities, and was 43 cm. long. The placenta was normal. Seven and a half hours after birth the child died without apparent cause. It had breathed and cried and seemed to be doing well.

This case illustrates the fallacy of basing positive evidence upon the condition of the fetal lung. Had this child not been born under careful observation, but had its body been found and an autopsy made, it might have been asserted that the child had never breathed. Its lungs sank in water, and not the slightest evidence of respiration was present. If the cause for this condition be sought, it must be ascribed to the congenital fragility of the capillaries in a premature child, born from a weak and ill-developed mother.

By reference to the physiology of respiration and from a study of the variations in intrathoracic pressure which the respiratory movement brings about, we find that the conditions are favorable for rupture of the capillaries of the lung. During forcible inspiration the air-passages are fully open, the lungs are greatly distended, and diastolic distention of the heart is present. There is also dilatation of the intrathoracic and intrapulmonary blood-vessels. The veins are more affected than the arteries. The heart-beat becomes small because the negative pressure in the thorax acts upon the thin walls of the auricles and impedes their contraction, so that but little blood flows into the ventricles. The conditions are such as to favor the accumulation of blood in the vena cava and pulmonary veins. Efforts at respiration during labor arise most frequently from interference with the circulation of the umbilical cord through pressure, or from direct pressure upon the body of the fetus. So soon as the fetus attempts to breathe in utero, the amniotic fluid is drawn into the nasal cavity, where it acts as a powerful excitant to the sensory fibers of the nasal mucous membrane and calls into action the inhibitory respiratory impulses. When we recall the pressure to which the child is often subjected in difficult birth, it can readily be seen that a child may be born in difficult spontaneous labor, breathe repeatedly, and then die, its lungs presenting a condition of apparent solidification which might lead to the belief that respiration had never occurred. The following cases illustrate the points just made:

CASE I.—A seven months' fetus. Mother admitted to the Maternity Department of the Jefferson Medical College Hospital in labor, the fetus dead. Transverse position, shoulder presentation, the child impacted in the pelvis in the form of a wedge. The arm was amputated at the shoulder, the wedge decomposed, and the fetus delivered. Autopsy by Dr. David Bevan showed both lungs deeply congested. No apparent hemorrhages. Blood flowed freely from the open pulmonary vessels. The walls of the ventricles were relaxed, the ventricles filled with blood, the coronary arteries turgid. Microscopic sections of the lungs

showed hemorrhage extending from the pleura into the lung tissue. In this instance the child had undoubtedly made respiratory movements during the long and fruitless labor of the mother before admission.

CASE II.—A female child, born at full term, asphyxiated, gasped feebly, breathed a few moments, and died. Its lungs were deeply congested, hemorrhages in the lungs similar to those in Case I. Its heart-walls were relaxed, the cavities filled with blood, and the coronary arteries distended. Its lungs sank immediately in water. Respiration, however, had actually occurred.

CASE III.—A full-term female child, delivered with great difficulty by the forceps. The head was severely bruised, and although respiratory movements were made, the child could not be permanently resuscitated. Artificial respiration was practised in this case. The left lung was completely collapsed. In the right the anterior free extremity of the middle lobe was partially inflated. Hemorrhages had occurred throughout the other portions of the lung.

CASE IV.—An infant dying shortly after a tedious spontaneous birth. Both lungs in a state of complete atelectasis. Numerous small hemorrhages beneath the pleura. Intense congestion. The abdomen contained a large quantity of blood-stained serous fluid. There were small hemorrhages beneath the two layers of the pleura. The heart-walls were relaxed. All the cavities contained blood. The coronary arteries were turgid.

The difference between the normal fetal lung and that in which hemorrhage has occurred is shown on Plate 1, the drawings for which were prepared by Dr. Bevan.

Spencer[1] has drawn attention to hemorrhages in the new-born accompanying pressure in birth, and describes hemorrhage beneath the pleura and into the lung substance, which he styles "pulmonary apoplexy." Such infants die very shortly after birth, and upon examination the lungs are not inflated.

It seems to me from this consideration that because portions of the fetal lung sink in water and are found to be practically solid, the inference cannot be drawn that the child has not performed the act of inspiration and expiration. Whether this respiratory movement occurred within the uterus during labor or just after the birth of the fetus cannot be told in a case where no one witnessed the birth. It is possible for a woman to be confined alone, or for her child to attempt to breathe for several minutes, and then perish without interference on the part of the mother. The lungs of such a child would show no signs of inflation, but multiple hemorrhage, as described, would be present.

On the other hand, the child may be still-born, and artificial respiration may partially distend its lungs with air. The method known as Schultze's, which consists in grasping the child's body, holding the head between the wrists of the operator, raising the body over the operator's head, allowing the trunk to flex, then bringing the body downward toward the floor, causes air to enter the respiratory tract. Other methods less vigorous are often effective. It might easily happen that a child, still-born, subjected to vigorous efforts to introduce air into the chest, might show a considerable inflation of the lungs, although it had never breathed.

From these considerations we may well ask the question, Upon what can a diagnosis of infanticide be made? We must reply that a very

[1] *Transactions of the Obstetrical Society of London*, vol. xxxiii.

PLATE I.

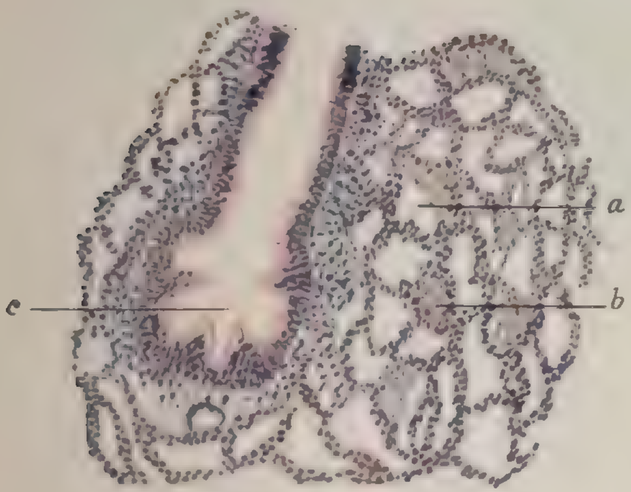


Fig. 1.

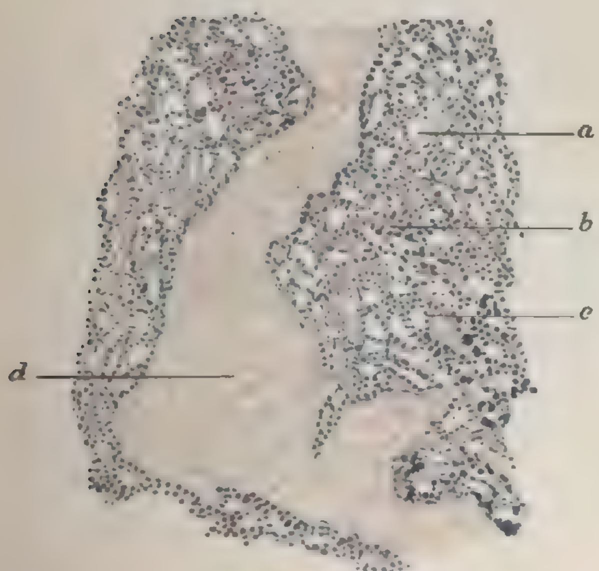


Fig. 2.

FIG. 1.—Human fetal lung, normal (Eye-piece 3; obj. Beck ½): *a*, Alveoli; *b*, inter-alveolar connective tissue; *c*, terminal bronchus.

FIG. 2.—Human fetal lung, showing effect of violent attempts at inspiration after rupture of the membranes, and complete stoppage of the circulation through the cord (Eye-piece 3; obj. Beck, ⅙): *a*, Alveoli; *b*, intra-alveolar connective tissue; *c*, dilated capillaries; *d*, area of hemorrhage.

careful study of all the circumstances of the case is necessary to form a correct judgment. It must not be forgotten that while the illegitimate child is most often destroyed, cases are on record where the legitimate infant has been sacrificed. As many illegitimate births occur while the mother is alone, the effort should be made so far as possible to ascertain the history of the labor. A point of considerable importance is the period of time elapsing between the rupture of the membranes and the birth of the infant. The discharge of the amniotic liquid exposes the child to more direct pressure, and hence would facilitate respiratory efforts on the part of the fetus and the injuries incident to birth-pressure. If it could be learned that the mother noticed a gush of liquid a long time before the actual expulsion of the child, we should thus ascertain that the conditions had been favorable for birth-pressure.

The relative size of the mother and the child is a factor of great importance. If the entire body of the child be found, it can be weighed and measured. The mother's pelvis can be measured, and if it is found that a considerable disproportion exists, that the labor was long and tedious, the membranes rupturing early, birth-pressure, as a factor in fetal injury, becomes a very important consideration. The body of such an infant might show hemorrhage upon the surface of the brain, which could lead an inexperienced observer to assert that a blow on the head had destroyed the child.

The respiratory tract and mouth of the child should be subjected to careful examination. It must be remembered that when a child does not breathe, the finger of the person present may be inserted into the mouth to remove the obstruction to respiration. If, however, a foreign body be found packed into the mouth, such as cloth, paper, or cotton, if finger-marks upon the throat are observed, and dissection reveals the effects of severe pressure beneath, it must be inferred that direct efforts at strangulation had been made. The meningeal hemorrhages following severe labor differ from those resulting from blows upon the head in the fact that in the former the scalp is less severely injured than in the latter; but it must not be forgotten that the child may be expelled spontaneously, fall head foremost upon the floor or ground, and be fatally injured without interference on the part of the mother.

Should it be found that the umbilical cord had been severed cleanly with a sharp instrument and had not been tied or crushed, the inference must be that an attempt had been made at infanticide through hemorrhage. On the other hand, if the cord were found to have been torn or crushed apart, infanticide could not be proved, because in spontaneous birth the cord may tear asunder and the fetus survive, and because the cord may be severed with a blunt instrument or crushed asunder without causing the death of the fetus.

If the fetus were found with its mouth and bronchi filled with blood, amniotic liquid, vaginal mucus, and fecal matter, the inference must be that it has lain face downward in discharges occurring from the mother's body at labor. It could not be shown if the mother were alone that

this had been intentional, and the only inference must be that she omitted to take up the child upon birth, or that she was in a physical condition in which she was unable to do so.

Infanticide by exposing the fetus to cold is exceedingly difficult to prove. Cases are on record where a fetus supposed still-born has been placed in a cold room, while nurses and doctors devoted their attention to the mother severely ill. To the surprise of all, the fetus has been found living after a considerable exposure of this sort.

Efforts at concealing the body of the fetus do not give positive proof of infanticide, as illustrated in the following case: The Obstetric Resident of the Jefferson Medical College Hospital was summoned to remove by ambulance to the Maternity Department a servant woman, illegitimately pregnant, and confined alone in her room at the house of her mistress. The mother was wrapped up and taken to the hospital as soon as possible, as the placenta had not been delivered. She asked to have taken with her her clothing contained in a small valise. Some hours after her admission the valise was opened to obtain fresh clothing for her, when the dead body of her child was found in the valise. Examination of the body of the child gave no evidence as to the cause of its death, nor could the mother be proved guilty of infanticide.

In concluding this section we must urge that each case be investigated thoroughly before a decision be based upon a single condition in the body of the fetus. The character of the mother, the circumstances under which she was confined, the presence or absence of an accomplice, the efforts to conceal the body of the child, or her readiness in acknowledging its birth, must all be considered. Her own physical condition, as well as the postmortem examination of the infant, should be taken into careful consideration.

IMPOTENCE AND STERILITY.

THE medicolegal relations of impotence and sterility are, in the largest measure, dependent upon the bearing of those conditions upon the validity of the marriage contract; but in some cases of alleged rape proof of impotence in the male may assume great legal importance, and we can also conceive cases of alleged sexual crime in which proof of sterility in the accused would be a demonstration of his innocence. Again, impotence or sterility in the male might be sufficient to prove absence of marital paternity. The importance of such possibilities in their relations to marriage and the family calls for examination of the various conditions that constitute or occasion lack of sexual power or absence of ability to fecundate on the part of the male, and of those conditions affecting the female which render fecundation impossible.

Sexual impotence means merely lack of power to perform normally the act of sexual conjugation; it may affect the female as well as the male.

Sterility is a term denoting, in general, failure to procreate under circumstances that ordinarily are attended by procreation. It is etymologically applicable only to the female, but it has come to be used to include conditions affecting the male in which the fecundative male elements are incapable of performing their functions. Thus, the application of sterility is restricted to cases in which failure to procreate or to conceive is due to causes independent of the act of sexual conjugation itself. Hence sterility does not imply impotence, nor impotence sterility, though both conditions may be combined in an individual.

IMPOTENCE.

The act of coitus is one in which many factors—physical, physiologic, and psychologic—enter, and these again differ as they are considered in the male or female; but since the male is the active agent in the act, his part is much the more important, and the one more open to functional derangement.

Impotence in the Male.—The part of the male in coitus consists of immission of the penis and emission of semen. The conditions that may interfere with the normal performance of coitus by the male, causing impotence in its narrow sense, may be divided into physical and psychic: *e. g.*, the obstacle to coitus may be some imperfection or defect in the penis or generative organs in general; or it may be some idea which prevents necessary erection or physiologic orgasm, thus effectually interfering with the initiation or completion of coitus.

The physical causes of impotence in the male are numerous, but they are, for the most part, included in deformities, congenital or acquired, of the penis. Defects of development of the penis vary in degree from slight anomalies of form and size to complete absence of the organ. The slight imperfections of form, when they do not interfere with erection, are no bar to the normal performance of coitus; but when they are more decided, and of such nature as to cause distortion of the organ in a state of erection, they may render immission impossible. Distortion of the penis in erection is due to some defect or malformation, natural or acquired, of the erectile tissue constituting the corpora cavernosa. Want of development of the corpora would render erection impossible; deficiency on one side would occasion a lateral deviation of the organ in erection; inflammatory changes or deposits in the erectile tissue interfere with uniform distention and thus cause distortion, the degree of which depends upon the seat, or seats, and the extent of the abnormality. Deposits in the corpora cavernosa may be congenital; but inflammatory changes, due to extension of inflammation from neighboring parts or to direct traumatism, are more frequently causes of deformities that result in sexual incapacity. In rare cases the penis is congenitally absent, while the other organs of generation are normally developed. In other cases the penis is so extremely small as to render coitus impossible. In striking contrast with the latter condition is enormous size of the organ, which as effectually prevents sexual intercourse. The penis is sometimes found attached to the scrotum; in such cases a surgical operation will usually render the individual sexually capable if the organ be otherwise normal. Cases of double penis with double urethra have been reported.

Other anomalies of development that may interfere with copulation are hypospadias and epispadias. The former condition is characterized by the orifice of the urethra being on the under surface of the penis instead of at the glans penis (Fig. 7); the latter, by the termination of the urethra being on the upper or dorsal aspect of the organ (Fig. 8). These anomalies are usually attended with more or less marked deformity of the organ as a whole. Whether an individual so affected is sexually capable or not depends upon the extent of general deformity, and upon the position of the urethral orifice. With slight general deformity intromission would be possible, but for the emission of seminal fluid to take place in the vagina the urethral orifice must be well toward the distal extremity of the organ. Even in some cases where the opening of the urethra is in the lower part of the glans penis itself, the consequent misdirection of the ejected seminal fluid prevents its reaching the os uteri, and the individual is practically impotent.

Phimosis may be of such nature and degree as to prevent a normal ejaculation of semen. Such a condition is usually open to surgical correction. Urethral stricture may cause impotence by preventing the ejection of semen entirely, or by allowing but a tardy and incomplete performance of the act.

Impotence caused by defect or disease of the testicles is to be classed

rather as sterility; for in such cases it is the male generative elements that are directly affected. When the generative glands are imperfectly developed, the functions of erection and orgasm will usually be imperfect, and such an individual might be classed as both impotent and sterile. In cases where the integrity of the testicles has been damaged by disease, the performance of coitus may still be possible, and the case is rather one of sterility than impotence.

Renal disease and affections of the prostate may lead to impotence, manifested in deficient erection and diminished sexual desire. A few diseases not directly implicating the genital organs are attended with impotence, permanent or temporary, depending upon the nature of the affection. Disease or injury of the nervous paths and centers which control the functions of erection and ejaculation may lead to permanent loss of sexual power. Certain general organic nervous affections, like locomotor ataxia and paretic dementia, often have a similar

FIG. 7.—Slight hypospadias (Hofmann).

FIG. 8.—Epispadias (Hofmann).

effect. The importance of these affections as causes of impotence from the present standpoint of discussion lies in the fact that the impotence may be manifested at an early stage of these diseases. In such cases there is commonly a simultaneous failure of sexual desire *per se*, showing functional or organic implication of the brain. Some general diseases affect more or less profoundly the sexual functions, but the duration of the resulting incapacity is usually only that of the cause. Neurasthenia is frequently attended by loss of sexual power. Diabetes mellitus sometimes induces a marked degree of impotence, probably through its tendency to induce changes in the nervous system. Chronic alcoholism may have a similar effect on the sexual functions, more or less permanent in character. Chronic morphinism, likewise, may temporarily or permanently diminish or destroy virility. It

seems probable that the excessive use of tobacco may have a similar effect.

Tumors and enlargements about the male genitals may effectually prevent the performance of coitus, thus making the individual practically, though not potentially, impotent. Such conditions are enormous hydrocele or hernia.

Another fertile source of male impotence lies in sexual excesses. Masturbation or overindulgence in natural sexual intercourse acts in two ways—through the direct weakening influence exerted on the nervous mechanism of the genitals, and the indirect influence exerted on the nervous system at large. Moderate indulgence in masturbation, like

FIG. 9.—Perineal hypospadias (Curtis).

moderate normal sexual indulgence, need have no detrimental physical effect, but it may, as a result of a psychic factor, be the direct cause of impotence of a psychic nature, discussed later. Sexual excess acts mainly in a way to exhaust the nervous energy of the genital centers, and to deteriorate the vital qualities of the specific reproductive elements of the sexual glands. Thus, a man so affected may become sterile while he is not yet impotent. Commonly such a condition is amenable to remedy, provided that a profound psychic change, leading to psychic impotence or sexual perversion, has not become firmly established. Instances of impotence due to masturbation and excesses in venery form a series of transitional cases lying between actual physical impotence on the one hand, and pure psychic impotence on the other. Indeed, it is rare for a case of impotence due to this cause to be devoid of a marked psychic factor as a contributing cause.

In many cases of impotence attributed to masturbation, that cause acts only through the mind. Impotence due to excessive indulgence in natural intercourse is usually physical first, with the subsequent addition of a psychic factor, serving but to intensify and prolong the primary physical condition. Psychic impotence, owing to its frequency and the comparative readiness with which it may be induced in persons apparently normal, is the most important variety of the affection.

Sexual desire is a function of the cerebral cortex. Through the activity of this psychic factor the lower spinal centers are excited to activity, and thus, under normal conditions, the genitals are stimulated to the performance of the generative functions. The genitals and their spinal centers are in a measure independent of the higher cerebral centers, as shown by the occurrence of erection and ejaculation when the nervous paths from the cerebrum have been altered by disease or traumatism, the change in the genitals being then a spinal reflex act. Ordinarily, erection is the result of psychic activity induced by impressions reaching the mind through the higher senses, and it is thus a psychic reflex act. But it is also true that the primary excitant of sexual desire may be excitation of the genitals locally, with the subsequent calling up of associated sexual ideas, which again react to intensify the activity of the spinal genital centers. This psychic element in sexual excitement is by far the most important factor in the sexual act. So important and essential is it, that without it, under normal conditions, erection is well-nigh impossible in mature individuals. Pathologic excitability of the spinal genital centers may induce erection, but without the intervention of the psychic factors it will be transient; or if persistent, attended by pain and discomfort which effectually overcome the element of pleasure, the most essential quality of normal sexual excitement.

The power of certain ideas to induce sexual excitement is counterbalanced by the power of certain other ideas to overcome and prevent it. These inhibitory ideas are the most essential factors in acquired psychic impotence, which includes the majority of cases of this nature. In another class of cases there is a want or loss of the usual ideas of a positive sexual nature. Cases are on record in which there was entire failure to develop sexual desire, notwithstanding normal development of genital organs and normal functional possibility. This congenital condition will be better understood after perusal of the subject of sexual perversion. This failure to develop normal sexual desire is often associated with other mental defects constituting the various degrees of idiocy and imbecility. It should be added, however, that there is no essential relation between intellectual defect and want of sexual desire. On the contrary, in many cases of congenital weak-mindedness there is a virtual or actual intensification of the psychic factor in sexuality, though it is likely, in such cases, to express itself in some perversion of the sexual instinct. This is the more probable in such cases, owing to the impossibility of the development of ideas of morality.

Loss of normal sexual desire *per se* may be induced by many con-

ditions. Thus, the masturbator finds greater pleasure in his act of self-abuse than in normal coitus, and at last the normal psychic excitants of the sexual impulse remain without influence on him. The man who indulges excessively in normal coitus is similarly open to the loss of pleasure in the act, finally resulting in a loss of power to respond to normal psychic stimuli of a sexual nature. Such an individual, if the lower genital centers are not exhausted, is prone to acquire some perverse manner of sexual gratification which has its charm for him in its novelty from a mental standpoint. Cases of this kind are mentioned under Sexual Perversion.

With advancing years, after full maturity, there is a normal lessening of intensity of sexual desire, usually accompanied by diminution in the functional power of the spinal centers, but this is not always the case; indeed, sexual desire may outlast physical virility, and thus induce sexual perversion. The duration of virility, however, is not to be measured by years, for sexual power, like all other functions, is a matter of individuality.

Acquired psychic impotence is also possible as a result of actual brain disease. Cerebral exhaustion, as seen in general neurasthenia, or from any cause, might prevent the psychic reflex. Organic disease implicating the brain or the downward conducting nervous paths may have a like and lasting effect.

Psychic impotence due to inhibitory ideas may be understood through examples of psychic inhibition in other relations. Thus, the inexperienced declaimer is commonly deprived of speech and memory when confronted by his audience. Try as he will, he cannot articulate a word of his well-learned speech; even the glands that provide moisture for the mouth refuse to perform their office. Some men of unusual modesty are incapable of micturition in the presence of strangers of their own sex. In this mental inhibition the idea of possible failure frequently is all there is to induce inability to perform some act, which would have been carried out had not the suggestion of failure arisen in some way. Under such circumstances the inhibitory idea acts more powerfully than the opposing positive idea, no matter how strongly accentuated by desire. A man of usual moral training is unable to perform coitus save in favoring circumstances of privacy. Any change of those conditions would effectually prevent erection; similarly, any interruption during the act would prevent its completion. Even when sexual excitement has reached an active degree under favoring circumstances the accidental interposition of unrelated ideas will ordinarily cause cessation of all sexual thought. These examples show how easily normal sexual thought is disturbed by intercurrent ideas, the ordinary cases of psychic impotence thus becoming clear.

Temporary impotence due to fear or modesty is not unusual in men who attempt coitus for the first time. Those who have practised masturbation and have had access to the evil suggestions of quack literature and medical works on sexual neurasthenia are often rendered psychically impotent by the thought and fear of impotence. Failure

under such circumstances may give rise to prolonged and even incurable impotence. The same is true in cases of men who have overindulged sexually, who, when they come to marry, have a doubt arise which renders them impotent with their wives. The power of habit and association in the performance of the sexual act may operate similarly. The circumstances attending the first sexual excitement and gratification in some individuals exercise a powerful effect on the subsequent activity of the sexual functions. The well-known case of the boy who was impotent for all women except those dressed like and resembling the girl with whom he had his first experience is a case in point. Men who have grown accustomed to the manners of prostitutes, when they marry may be impotent for lack of the long-accustomed stimulus which the wives, in their natural ignorance and modesty and comparative coldness, cannot offer. The habitual masturbator may be psychically impotent because he misses, in attempt at intercourse, the pleasure he finds in his habit.

Another form of psychic impotence is that due to overintensity of sexual excitement, as a result of which the sexual orgasm occurs before immission is possible. This form is apt to affect the inexperienced, and in some cases it becomes so confirmed as to render the individual practically impotent. Pathologic hyperexcitability of the sexual centers, as a stage in sexual exhaustion, may lead to the same result.

Impotence in the female may be due to a great variety of anatomic conditions which act to prevent the proper performance of coitus: *e. g.*, absence of vagina, grave deformities of the pelvis, adhesion of the labia, imperforate hymen, too dense and resisting hymen, atresia of the vagina, etc. Tumors about the genitals may have a similar effect.

Fissures and ulcers at the introitus vaginæ, and even at the anus, are fertile causes of the condition called vaginismus, in which, on an attempt at coitus, the constrictor muscle of the vagina becomes so firmly contracted that *immissio penis* is impossible. In some of these cases pain is the essential factor; but in others, fear or an idea is the disturbing element that renders coitus impossible, and the woman might properly be called psychically impotent.

The woman's part in the sexual act is so essentially passive that it is much less open to disturbance than that of the man. Many women are, doubtless, never truly excited sexually, and never experience a complete sexual orgasm. A woman may have such a mental aversion to the sexual act as to prevent its normal completion on her part, and such a state of mind might lead to a persistent refusal to indulge in it—a state of true psychic impotence. Passive submission, under such circumstances, might suffice to satisfy the husband, but where the aversion on the part of the wife becomes so excessive and troublesome as to constitute a bar to ordinary indulgence, it might, like serious anatomic obstacles, constitute a ground for divorce.

STERILITY.

Sterility in the male must be dependent upon some anomaly of the testicles or of the seminal passages. Absence of the testicles or of the seminal canals would, in either case, cause complete sterility. Congenital absence of the testes is very infrequent. Castration after the age of puberty would not necessarily cause complete sterility until after the seminal passages had been emptied of all contained spermatozoa. It should be remembered that castration in itself does not necessarily remove the possibility of erection and coitus. The loss of one testicle need not interfere with procreative power if the other be normal. Absence of the testicles from the scrotum is in itself no sign of sterility, since in rare cases they do not descend.

The function of the testicles is the secretion of semen and spermatozoa, and the semen depends upon the presence of the spermatozoa for its fecundative power. This function begins with the occurrence of puberty. The age at which puberty is established varies somewhat with race, climate, and education. With us it occurs from the fourteenth to the eighteenth year. Sexual development is of gradual growth, as is evidenced by the gradual development of the changes of general form, growth of beard and genital hair, change of voice, etc. Premature excitation of the sexual organs and the cultivation of sexual thoughts have an undoubted influence to hasten sexual development. Age is much less to be relied upon as an index of sexual maturity than other conditions manifest in the individual. Cases have been reported of sexual maturity before the age of twelve. Erection occurs in very young boys, and it is doubtless true that erection and ejaculation of semen occur at an age when the semen has not yet attained fecundative power.

Just as sexual desire diminishes in old age, so the fecundative capacity decreases; but it is impossible to give any fixed period at which the latter ceases. Men of very advanced years are sometimes capable of procreation.

Congenital defects of the testicles are not of frequent occurrence; but in some cases the testicles remain undeveloped. Such anomalies are often associated with other bodily imperfections of development and with mental deficiency. Atrophy of the testes occurs as a senile condition, but it frequently is manifested earlier in life as a result of various conditions. Excess in sexual intercourse and masturbation are capable of inducing it. It is also frequently the result of inflammatory processes affecting the testicles and epididymis; and it may result from extraneous pressure, as in varicocele. Atrophy of the prostate gland occurs with atrophy of the testicles, a circumstance that is now taken advantage of in enlargement of the prostate, castration being followed by atrophy of the latter. Injury or disease of the spermatic nerve in its origin or course entails progressive atrophy of the corresponding testicle. Alcohol and morphin, when long indulged in, may cause sterility. Fatty degeneration of the testicles is likely to occur in very

stout persons, just as a similar change affects other organs in such persons. Probably in progressive atrophy of the testicles the secretion of the spermatozoa diminishes and ceases before there is any decided change in the secretion of the semen. Such a person might thus become sterile long before becoming incapable of erection and ejaculation. Inflammation of the spermatic ducts and the epididymis of gonorrheal origin is, perhaps, the most frequent cause of impotence. The canal becomes obliterated at same point, and the fluid ejaculated thereafter is secreted along the remaining pervious portion of the ducts, and contains no spermatozoa. To induce sterility, both ducts, of course, must be impervious. After coitus has been often repeated, the semen last ejected may be poor or absolutely wanting in spermatozoa—a temporary condition. Doubtless certain general diseases of an exhausting nature have a similar temporary or permanent effect. Rare must be the cases in which, in an absence of disease, spermatozoa are absent congenitally.

Absence of the vas deferens as a congenital defect must be very rare. As already indicated, it is frequently obliterated in inflammatory processes. This in itself does not cause orchitic atrophy. The ejaculatory duct is liable to injury in the operation for stone, and cases of sterility after perineal lithotomy are on record. The power to ejaculate semen may thus be lost, though coitus (immission) may be still possible.

Sterility in the female is by far more frequent than in the male. A woman's years of fruitful sexual activity are much more definitely fixed than a man's. Capability of conception begins with the occurrence of menstruation, and continues during the time of the periodic recurrence of that function. Menstruation begins usually from the twelfth to the eighteenth year, depending on individual development. It has been known to begin in infancy and continue regularly to recur. It is presumable that in such cases the capability of conception has not undergone a corresponding precocious development, but instances of impregnation at a very early age—eight years—are well known; and the development of ripe Graafian follicles takes place long before the attainment of puberty. It is important to remember that female sexual maturity may occur before menstruation appears; indeed, menstruation may be long delayed, and occur for the first time after one or more pregnancies, or not at all, though there be no other sexual anomaly. Capability of conception usually ceases between the ages of forty and fifty. As a rule, the cessation of menstruation is a sign of loss of power to conceive, but it does not in itself imply that power to conceive is gone, for in rare instances women have conceived one or more years after the menopause. It is rare for women to conceive after the age of forty-five, but well-authenticated cases are on record where the age was above fifty, one at fifty-five.

Sterility in the female may result from a great variety of pathologic processes and conditions. Congenital absence or defect of the ovaries occurs, but usually with other genital defects, as in certain cases of so-called hermaphroditism. Acquired absence, as a result of surgical operation, is, unfortunately, becoming too frequent. Both ovaries must

be entirely wanting to induce sterility from this cause, for any portion of them is sufficient to allow conception. Ovarian tumors may or may not cause sterility, depending upon complete or incomplete destruction of the organs. Other diseases of the ovaries and tubes may cause sterility, especially double salpingitis and pelvic inflammatory processes. Congenital absence or defect of the uterus precludes conception. In some cases of this kind the condition of the vagina still permits coitus. Versions and flexions of a uterus otherwise normal are very frequent causes of sterility, probably owing to pathologic processes so common to uteri in abnormal positions. While fibroids and carcinomata of the uterus are ordinarily obstacles to conception, they are not always so. The various forms of uterine inflammation, deformities of the neck, and leukorrhea are fertile causes of sterility, but they are not necessarily followed by it. Narrowness of the vagina, even almost complete closure of it at some point in its course, is not invariably a cause of sterility. This follows from the fact that complete *immissio penis* is not in itself essential if there be a way for the spermatozoa to find an entrance from the external genitals. Vesicovaginal fistula may form an insurmountable obstacle to conception.

While a woman may be capable of coitus and conception, she may still be unable, as a result of disease or deformity, of bearing a child. Such a condition might, like those previously considered, be a cause of medicolegal inquiry.

Questions that arise in connection with impotence and sterility are concerned with the duration of the condition previous to the time of its discovery and its probable continued duration. In some cases, as may be seen from the foregoing, it is easy to answer these questions; in others, only a guarded opinion and prognosis can be given. Both must be founded upon careful observation of the case and a wide experience with allied cases.

RAPE.

RAPE is carnal knowledge of a woman by a man, accomplished by force against the will and without the consent of the woman. Carnal knowledge is constituted by penetration, even in the least degree, of the female genitals by the male organ. That this shall constitute rape, the facts of force, resistance, and absence of consent are legally essential. The force used must have been sufficient to accomplish the purpose, but it need not be actual physical force; moral force, quite as compelling as physical force, may be exerted by means of threats of physical violence or other manner of injury, and this need not be directed immediately against the victim, as where threats against husband, children, or other relatives compel submission. Intercourse accomplished by means of deception, where there has been neither threat nor purpose to use violence or force of any kind, is not rape in the eyes of the law.

Resistance is the most immediate expression of the act being against the will of the female. This must have been manifested positively by her, or she must have been in a physical or mental condition precluding its expression, to make an act of intercourse with her rape. Absence of resistance as a result of physical incapacity, narcotism, or mental disease is not construed to change the character of an unlawful act of intercourse.

Lack of consent is construed generally to imply that the act is against the will, and the legal treatment of "consent" is thus of primary importance in any consideration of rape. The law fixes the capacity of the female to give consent more or less definitely by her age. Intercourse with a female who has not attained the age of consent is, in general, felony; under some enactments it is a misdemeanor; where the child is between the ages of ten and twelve, and gives consent, it is a misdemeanor. The age of consent in the majority of the middle states is fourteen; in New York, it is sixteen. Beyond the age of consent, where consent to intercourse is given, even with the greatest reluctance, there can be no question of rape.

The capacity of the male to commit rape is also affected by age and other conditions. The male must be physically capable of intercourse. In English law the perpetrator must have attained the age of fourteen, but in the middle states some courts have held that age does not, in itself, furnish proof of incapacity. Proof of impotence constitutes no defense for *attempt* to commit rape.

The victim of rape is a competent witness, but it is necessary to corroborate her testimony when guilt is denied. The medical witness will, in many cases of alleged rape, be called upon to give evidence touching

the question whether penetration has taken place or not, and whether force was used. As already indicated, the fact of penetration is the only essential part of intercourse that needs to be established as an element in rape.

Examination of the female genitals is the only means of determining physically whether penetration has taken place or not, and it is, therefore, necessary to consider the changes induced in the female generative organs by coitus.

In their virgin state the female genitals present the following characteristics: the labia majora are firm, large, and well rounded, their median surfaces lying in close contact and covering completely the labia minora, the mucous membrane of the latter being of a bright rosy tint. The vestibule is narrow; the hymen is intact; and the vagina is narrow, presenting marked folds of its mucous membrane. The condition of the labia majora depends far more upon the state of general nutrition, age, and position than upon anything else. Coitus, frequently repeated, will cause no change of the virgin state of the labia if the female is young and well nourished. As a woman grows older the subcutaneous fat commonly diminishes, and a virgin under such circumstances would present more or less marked separation of the labia majora. The same is true in women emaciated from any cause. Separation of the thighs is normally attended by more or less separation of the labia. The rosy tint of the labia minora depends upon the protection given by the labia majora; where they remain uncovered by separation of the latter, the mucous membrane comes to resemble epidermis, in being brownish and dry. Thus the condition of the labia and nymphæ can give no unequivocal evidence of the existence or absence of virginity.

The hymen, owing to its structure and position, is capable of giving more decisive evidence of virginity or defloration. The hymen from the earliest times has been regarded as the sign of virginity, but more careful study of its variations and the alterations to which it is subject has markedly modified earlier views. It was long regarded as a fold of mucous membrane developed at the orifice of the vagina, but it is the representative in the female of the corpus spongiosum, and thus represents an organ modified by arrest of development. This morphologic identification of the hymen accounts for its constancy—there is no authentic case of its absence—and the numerous variations in form to which it is subject. It makes its appearance soon after the fourth month of fetal life, in the form of two folds at the opening of the urogenital sinus, which later fuse in the median line, save at the hymenal opening. The primitive folds of the hymen extend forward to embrace the clitoris, and their union in the median line of the vestibule forms the frenum masculinum. The folds of the infantile hymen may be prominent, while the vestibule is relatively deep-seated, and at this early age the hymen may be mistaken for the nymphæ. When the hymen is deep-seated, a persistence of the usual infantile condition, the erroneous conclusion of its absence may be drawn.

Commonly the fully developed hymen is thin and membranous, with an opening leading to the vagina; but there are numerous variations from this form. The hymen is sometimes thick and fleshy, offering an insurmountable obstacle to coitus; in other cases it is so thin and elastic that coitus and even labor are possible without its rupture.

The bearing of these possible states of the hymen upon the question whether coitus has taken place or not is obvious: an intact hymen, strong and unyielding, with a small hymenal orifice, would be conclusive evidence that complete penetration had not been had; an intact elastic hymen would not afford unequivocal negative evidence.

In childhood the hymen frequently has the labiated form, a continuance of the fetal condition (Fig. 10). Fusion of its folds sooner or later gives rise to the ordinary annular form (Fig. 11). The common annular hymen varies in shape with variation of the size and position of the

Fig. 10.—Hymen with linear opening (Tardieu).

Fig. 11.—Annular hymen (Tardieu).

orifice. When the opening is large, the hymen may be nothing more than a membranous fringe at the vaginal entrance. Usually the opening of the hymen lies anteriorly to the center of the entrance of the vagina, when the hymen will be more or less crescentic if the opening extend forward to the anterior edge of the vaginal wall (Fig. 12). The opening may be circular, but it is more often oval, with the long diameter extending anteroposteriorly. The size of the hymenal orifice varies within wide limits: it may be so large that there is no narrowing of the *introitus vaginæ*; or it may be so narrow that it will allow the passage of only a small sound.

Imperforate hymen is a rare condition; *atresia vaginæ* may be mistaken for it. When the opening of the hymen is long and narrow, the hymen presents two symmetric halves, and is called labiated, as in its early form. Varieties of form due to peculiarities of the free edge of the hymen have received particular designations, the most striking being the fimbriated hymen, characterized by the edge presenting numer-

ous minute processes giving the appearance of fringe (Fig. 13). This fringed condition is but a marked development of notches that are present in many cases. These notches, owing to the fact that they may be readily mistaken for evidences of rupture, are of much medicolegal

FIG. 12.—Crescentic hymen.

FIG. 13.—Fimbriate hymen intact.

importance. Such indentations may be situated almost anywhere on the edge, but they usually occur symmetrically and anteriorly. They may be of any depth, even extending to the vaginal wall and dividing the hymen into valves (Fig. 14). To avoid mistaking such natural irregularities for artificial tears, all that is necessary is to note the ab-

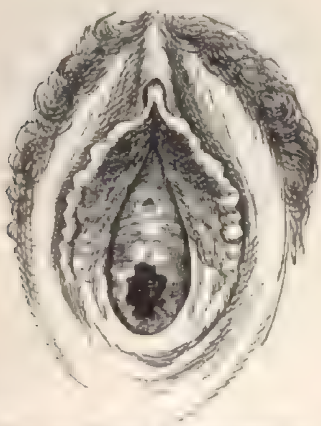


FIG. 14.—Circular hymen presenting natural notches.

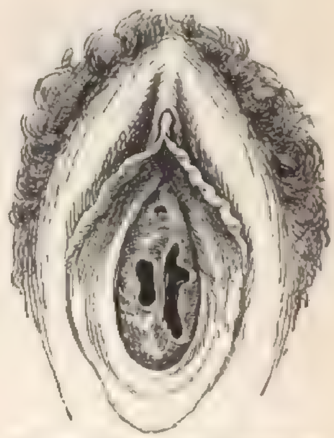


FIG. 15.—Hymen presenting a septum and unequal openings.

sence of cicatricial tissue and the integrity of the mucous membrane at the suspected points.

The normal single opening of the hymen may be found divided by a septum into two equal or unequal apertures (Fig. 15). The septum

usually extends anteroposteriorly, but its direction may be oblique. There may be, however, but one lateral opening. The remnant or rudiment of a septum is seen rarely as a long slender process with a pos-

FIG. 16.—Hymen with posterior rudimentary septum.

FIG. 17.—Hymen with posterior rudimentary septum.

terior attachment (Figs. 16 and 17). The cribriform hymen is a rare form, presenting numerous minute openings.

It is important to note that the condition of the hymen varies with

FIG. 18.—Purse-like hymen undistended.

FIG. 19.—Crescentic hymen presenting two lateral lacerations and posterior valve.

variation in the position of parts about it. It does not present as a tense membrane. With the genitals in their usual position, the hymen is folded on itself (Fig. 18). The crescentic hymen has a median antero-

posterior fold; the edge of the circular hymen is folded into a cone extending outward. In adults, wide separation of the thighs and labia makes the hymen tense, but in children, owing to the greater extent of the hymen as compared with the *introitus vaginæ*, it ordinarily remains lax under like circumstances.

Strong presumptive proof of virginity is presented by an intact hymen, but its value must be decided by consideration of the peculiarities of the hymen itself—its elasticity, the size of the opening, etc. It is to be remembered that "penetration" does not imply penetration beyond or even to the hymen. It must be remembered that gradual dilatation of the hymenal opening is possible.

Usually the hymen is torn more or less in the first act of coitus. Of course, the nature of the hymen will have much to do with the peculiarities of the resulting lacerations. Such tears commonly start from the edge and extend to the vaginal wall. The labiated hymen is

Fig. 20.—Circular hymen torn in several places.

Fig. 21.—A ruptured fimbriate hymen.

ruptured posteriorly in the median line; the crescentic hymen tears symmetrically on each side, leaving a posterior median valve (Fig. 19); the circular hymen commonly tears in several places (Fig. 20). Much will depend upon the comparative weakness of the parts of the hymen in determining the direction and character of lacerations. The hymen divided by a septum may be ruptured on only one side.

Rupture of the hymen is quite invariably accompanied by hemorrhage; it has even been known to occur with such profusion as to cause grave alarm and even endanger life.

It cannot be difficult to recognize a recent rupture of the hymen, the unhealed edges of the laceration and the inflammatory swelling of the parts remaining as conclusive evidence. Slight lacerations of the hymen heal in two or three days; more severe tears may require much longer before repair is complete. At any time before healing has taken place and inflammatory swelling has subsided the diagnosis of rupture is easy;

but it may be difficult at a later period. Much will depend upon the extent of the lacerations and the consequent formation of cicatricial tissue. In cases of slight rupture at this period special care must be taken to distinguish between presumptive tears and natural notches. Examination of the hymen for possible evidence of rupture should be conducted with care to avoid inflicting injury that may subsequently be mistaken for a previously existing tear.

Rupture of the hymen, since it may be due to many causes, is not conclusive evidence that coitus has taken place; but it is rarely possible for the hymen to be injured without penetration of the genitals to some extent. Forcible separation of the thighs has been alleged as a cause of rupture of the hymen, but such an event is hardly possible, especially in children, unless neighboring parts are simultaneously implicated in the laceration. Isolated rupture of the hymen by a fall on the perineum, unless impact were on a projecting object, would be a rare accident. The practice of masturbation may lead to injury simulating evidence of coitus, but this must be infrequent in children; for in them the irritative rubbing is directed quite exclusively to the clitoris and inner surface of the labia. Persistent pruritus in children is more likely to lead to injury of the hymen than the common habit of masturbation. Children frequently attempt to pass objects into the urethra, but usually leave the entrance of the vagina untouched. Scars on the hymen may be the result of diseases like small-pox, noma, diphtheric inflammation, and venereal diseases.

The first act of coitus may inflict injury involving other parts as well as the hymen. The delicate structure of the fourchet renders it liable to such injury; and even rupture of the perineum and laceration of the nymphæ are reported from this cause. Such an event in the case of a mature female must be very rare, but in a child it would seem more easily effected. The presence of such extensive lacerations at once raises the question of the manner of their infliction; for the limited power of the penis to overcome resistance would indicate that they were due rather to the use of the finger or other more rigid object. Penetration of the finger may cause rupture of the hymen like that due to coitus, but it is also capable of inflicting much more serious and extensive injury.

The vaginal walls and mucous membrane present characteristic folds in the virgin, and this condition is altered only by repeated acts of coitus. In children, if the vagina has been distended, there may be evidence of it in the dilated condition of the passage.

The presence of semen in or about the genitals of an alleged victim of rape is incontestable evidence of sexual contact. This proof must be sought in the secretions of the parts, and in any dried secretions obtainable from the genitals or clothing. The characteristic element of semen is the spermatozoon, which may be found as readily in dried secretions as in fluid. The spermatozoa retain their form indefinitely in seminal stains that have been dried. Skilful examination for them with the microscope is, of course, necessary.

The existence of venereal disease in the person of an alleged victim of rape is often of great importance as evidence of sexual contact. In such cases the medical witness has need of great caution in diagnosis, as well as of great care in giving an opinion. The true venereal affections are easily simulated by more innocent conditions, at least upon superficial examination. Gonorrhea may be positively diagnosed by the discovery of the gonococci, but failure to find them in the secretions is not a demonstration of the non-venereal nature of a genital catarrh, for in the later stages of the disease the gonococci may be found only with great difficulty or not at all. In acute gonorrhea the gonococci are found in the vaginal secretions and in the moisture on the inner surface of the labia; in the chronic stages they may be found in the cervical canal and in the urethra. Thus, failure to find gonococci in the discharges accompanying an acute genital catarrh would support the presumption that the disease was of a non-venereal nature. It should be remembered that existing venereal disease is not direct proof of sexual contact, no matter how strong it may be as presumptive evidence of it. The genitals may become infected in many other ways. Children have developed gonorrhea from using articles infected by others. Genital infection is possible from infected hands, directly or indirectly. Syphilis may be communicated in various ways aside from sexual contact, especially by kissing. Simple genital catarrh is not uncommon in female children and adults; it is of frequent occurrence as a result of the first coitus, especially when there was preëxisting leukorrhea. Suits for divorce have been instituted on the erroneous assumption that the inflammation thus arising was specific. Females that have been raped may present such an acute inflammation, which, if mistaken for gonorrhea, may give opportunity for a false defense in that the ravisher may prove himself devoid of disease. Other common causes of mucopurulent genital catarrh in females are disordered menstruation, masturbation, skin diseases, oxyuris, and any general enfeebled state of health. Genital catarrh is sometimes epidemic, and the genitals are subject to diphtheric inflammatory processes.

Simple ulcerative processes affecting the female genitals are usually easily differentiated from those of a specific nature; but syphilitic sores are not always indurated, and simple ulcers may be indurated and simulate the hard chancre. Soft chancre can be diagnosticated by auto-inoculation; and it may be necessary to distinguish it from herpes. The genitals of female children may be affected with ulcerative processes after scarlet fever, typhoid fever, and measles.

It will be seen that there are indefinite possibilities of cases arising in which great care in diagnosis must be exercised. Even the incubation and course of venereal affections become important, since it may be necessary to give an opinion affected by the time of alleged infection and the stage of a malady found at the time of examination. The importance of these points is further emphasized by the frequency of false accusations, to be discussed hereafter.

Examination of the accused may be of equal value for the establish-

ment of guilt and innocence, but it can be practised only with the consent of the accused. It is especially important in respect to venereal disease, when a similar affection is found in both victim and ravisher. Acute gonorrhea in the male is certainly diagnosticated by the microscope, but a gleet more easily escapes detection, and precaution should be taken accordingly by the examiner. A gleet is less infectious than an acute gonorrhea; on the other hand, the genitals of virgins and female children are more readily infected than those of women that have been accustomed to coitus. Sexual contact with a diseased male is not always followed by infection of the female; nevertheless, presence of the disease in one and not in the other of the two individuals forms strong presumptive proof that they have not had sexual contact.

When the accusing female is one that has frequently practised coitus, the single act of normal coitus will leave no anatomic evidence behind; in such cases, however, there is not infrequently evidence of the force used about the genitals or elsewhere on the person of the victim.

Physical evidence of force used in the accomplishment of rape affords tangible proof that the act has been accomplished against the will of the female, and the medical witness may be called upon to interpret such evidence. Severe or serious injuries resulting from blows, choking, or brutal physical violence are a direct measure of force used; slight injuries should be viewed also in the light of the possibility of their having been self-inflicted, especially with reference to their situation on the person of the victim. Unless the victim is examined soon after the violence has been used, slighter marks of it, like bruises and scratches, may have disappeared; wounds and extensive abrasions are in evidence longer. Such evidences of the use of force may be found in almost any situation on the person, but the neck, thighs, arms, and external genitals are most frequently the seat of injury. Natural pigmentations have been mistaken for suggillations. Injuries are always to be considered with relation to their condition and the alleged time of their infliction.

Signs of violence found on the person of the female speak also for the degree of resistance she has offered to the sexual approach. The law demands that a woman exert all the resistance of which she is capable. In many cases the signs of violence on her person and on the person of the ravisher show that resistance has been strenuous and quite adequate to her physical strength; but absence of such evidence of resistance must not be construed as conclusive proof that the woman has not offered all the resistance of which she was capable. The physical condition of the victim is not alone to be considered; it should be remembered that nothing is so likely to cause temporary psychic paralysis in a woman as a violent and unexpected sexual approach. Fear of intent or threat to inflict bodily injury is a potent source of failure to use strenuous physical resistance. Attempts to gain help by outcry are means of resistance, but even failure to make use of this is not evidence of failure to resist when fear or psychic paralysis intervenes to prevent the cry of alarm. Lack of proportion in physical

strength is usually in favor of the man, but not always; and even where the woman is the stronger, the mental factor of feminine timidity and possible surprise should be considered. The frequency of false accusations makes it necessary, however, to keep in mind the bearing of relative physical strength in the two persons.

Signs of violence on the person of the ravisher may be found in the form of scratches, bruises, and wounds made by bites; the male genitals often present signs of slight or even severe injury. Such findings are indicative of the degree of resistance offered by the victim.

When the possible power of resistance is overcome by fear aroused by threats, no matter what their nature, the moral force thus employed is equivalent to physical compulsion. The possibility of resistance may be precluded by physical infirmity or by mental conditions more thoroughly discussed in a subsequent paragraph. When fraud has been the means of accomplishing coitus, the act cannot legally be construed as rape unless it can be shown that there was intention to use force, which the success of the fraud made unnecessary. Thus, for example, coitus accomplished under cover of darkness, the woman mistaking the ravisher for her husband, is not rape. This seems logically inconsistent; for commonly unlawful intercourse with a woman narcotized by drugs or intoxicants is rape; and it would seem that deception, however practised, since it precludes the action of sound judgment as readily as do narcotics, should be viewed in the same light.

Unlawful connection with a woman had while she is in a state of unconsciousness is commonly rape; it matters not what the cause of the unconsciousness may be. It is possible for coitus to be had with a woman while she is asleep; but it would seem necessary that she be in a state of profound sleep, and a woman accustomed to sexual contact. The first act of coitus would certainly arouse a woman from any ordinary slumber. Sleep, a favoring position, and the absence of impeding garments might so facilitate the initiation of a sexual approach that, when the woman had become aroused, her resistance would prove futile. Pathologic or artificial states of unconsciousness are commonly deeper than normal sleep, and are the more readily taken advantage of for a sexual purpose. This is commonly interpreted as rape. Such a condition may be induced with a view of using it to facilitate sexual approach. When a woman has been induced to take some common form of alcohol, and her subsequent state of intoxication has been used to facilitate sexual congress, the case is not so simple as to constitute rape without other consideration. If in such a state she offer resistance, it is rape; if she give consent, unconsciousness of acquiescence must be proved to make the crime rape. Under such circumstances it would be assumed that an adult female was aware of the effects of the ordinary forms of alcohol, and her consent to take that which lowers moral tone would help to lessen the responsibility of the accused. In the case of a young and ignorant girl, her indulgence in alcohol and subsequent consent in a state of intoxication should not lessen the degree of responsibility of her ravisher.

Any of the narcotics, but especially chloroform, ether, nitrous oxid, opium and its alkaloids, and chloral hydrate, might be administered with a sexual object in view, but there are only a few cases in which this has been actually done. In most recorded cases where such agents have played a part in rape the narcotic has been given for some other purpose, and the unconscious state has offered a favorable opportunity. It is possible to chloroform a sleeping person without breaking the slumber, but it requires great care and skill, and the chances of failure and success are about equal even under most favorable circumstances. Other states of temporary unconsciousness might be used for sexual purposes; syncope, epileptic unconsciousness, cataleptic and hysteric conditions, and the hypnotic state may be mentioned in particular. However, accusations of rape based on testimony related to such states of mind should be most carefully scrutinized, since such cases have been repeatedly shown to be instances of intentional or delusional false accusation.

The frequency of false accusations of rape is notorious, and demands particular discussion. The possibility of such a state of the case should never be left out of account in any instance. Motives of such false charges vary; they may be made with the purpose of revenge, blackmail, or even ignorantly; or they may be the result of hallucinatory or delusional ideas. When false accusations are made from motives of revenge or for the purpose of blackmail, attempts often are made to manufacture evidence of a physical kind corroborative of the charge. The female may inflict injuries on herself to counterfeit signs of violence. Such injuries are usually trivial in nature, and are situated on parts of the person easily accessible to her hands—*i. e.*, the limbs and genitals. Such self-inflicted injuries are more likely to be present as abrasions and scratches than as bruises. In cases where there are signs of violence, care is always to be exercised to study them with relation to their condition and the time of their alleged infliction.

False accusations, made by designing women for the purpose of blackmail, are on record. After exciting genital inflammation in their children, they have made this the basis of complaint. Ignorance of the fact that young children are often subject to acute genital catarrh of an innocent nature has often led to trials for rape, on the assumption that a child was suffering with specific venereal disease. Men who have been innocently associated with such a child are the easy victims of such a combination of accidental disease and common ignorance and suspicion.

Another class of false accusations is based on the complaint that some narcotic, like chloroform, was used to produce unconsciousness. Such a charge should be circumstantially investigated to ascertain whether the alleged facts pertaining to the administration and action of the drug in question are in accord with known characteristics of its action; or whether, in case the agent is not known, they correspond with the action of any known drug.

False accusations of rape committed during pathologic states of unconsciousness demand most careful study; for such mental states are

often followed by hallucinatory and delusional ideas that may have had origin in dreamy ideas present in the unconscious state. In these and allied cases, in order to render the accusations convincing, they should be substantiated by objective evidence of the most unequivocal kind. The instant a plaintiff attempts to describe events that took place while she was unconscious, the testimony is invalidated; but it is to be remembered that even so-called states of epileptic unconsciousness may leave behind a dreamy summary of events that took place during its continuance.

There is no doubt that the hypnotic state might be induced and used for a sexual purpose. Accusations of rape based on such a condition, however, call for the greatest caution in the expression of an opinion by the medical witness. States of unconsciousness may be induced by hypnotic procedures and suggestions, or the subject may be placed in a state in which she is devoid and incapable of will, entirely controlled by the operator, but perfectly conscious of what takes place. It is to be remembered that the hysteric are very amenable to hypnosis; that the hypnotic state of mind is preëminently hallucinatory, and, therefore, a favoring soil for the growth of lasting delusions; that the hypnotic state of unconsciousness is abnormal in itself; therefore testimony derived from such mental states should be accepted only with a full understanding of these facts. The celebrated trial of the hypnotist Czinski, in 1895, who was convicted of a sexual crime, as published by Enke, of Stuttgart, contains a lengthy and instructive practical review of the opinions of authorities on this subject.

Autohypnotic and cataleptic states are to be viewed in a light like that essential to an understanding of hypnosis induced by a second person.

Physicians and dentists are especially open to the danger of being subjected to false accusations by females; commonly such complaints are based upon a state of unconsciousness existing during the action of some anesthetic. The most frequent form of accusation is that, during the state of unconsciousness, while deprived of all power of will or resistance, the assault was made. Such a statement should be scrutinized carefully and corroborated by other evidence, if possible, before it is given decisive weight; for there are cases on record where such complaints were shown to be hallucinatory by the testimony of the most disinterested witnesses—and even by parents of the girl—who were present at the time of the alleged assault.

False accusations of rape are often made by women while in insane conditions; the possibility of such an event must never be allowed to outweigh the necessity for caution in crediting statements of a sexual character made by an insane female. Objective evidence should be required.

When a considerable period has elapsed between the date of the alleged assault and the lodging of the complaint, unless the delay is obviously the result of innocence, shame, or ignorance, the possibility of a motive of revenge or blackmail should always be considered.

Physicians, perhaps, more than men of any other class, are open to false accusations of a sexual nature, made by designing females with the purpose of blackmail; this possibility is to be remembered in such cases.

When an idiot, imbecile, or insane person is the subject of a sexual assault, the crime is commonly rape, for, aside from resistance and force, such persons are incapable of giving legal consent; but it would also be essential for the prosecution, in such a case, to prove that the ravisher was aware of the irresponsibility of the female.

Since the age of consent, in the case of youthful females, is the pivotal point upon which the crime of rape turns when coitus has been accomplished without force, it would seem that the actual and apparent age should be considered in case the offender yielded to solicitations by one apparently beyond the age of consent.

It is a sad fact that the most frequent sexual crime is assault of female children ranging in age from babyhood to early girlhood. Such assaults in Europe are sometimes due to the belief that intercourse with a virgin (child) cures gonorrhea; sometimes to the hope that childish ignorance and weakness will make resistance slight or success easy; sometimes to fears of impotence on the part of the male, who hesitates to make an attempt with an adult through fear of ridicule (the inexperienced and the senile). It is possible that a debauchee might attempt to find lost pleasure by such a crime. A true sexual perversion might express itself in a preference for children. Of course, in many cases children are the accidental victims of proximity to a man inflamed by inordinate passion under favoring circumstances. In cases of rape of very young children the sexual contact without serious injury can consist of nothing more than vulvar penetration. Where the genitals are found wounded or torn after such an attack, the lesions are usually the result primarily of attempted dilatation by means of the finger or other hard object. But the genital passage of a child is capable of being greatly dilated by repeated gentle efforts; and such disproportionate dilatation, without lesions, should lend color to the suspicion that a child presenting such an anomaly had been improperly approached.

There are secondary consequences of rape that may raise important legal questions. Impregnation entails additional liability in law. Aside from the secondary consequences of the venereal affections, injury inflicted in a rape may lead to serious or even fatal secondary results. Death may speedily result from shock, hemorrhage, or grave injury; or it may follow later from sepsis or hemorrhage into the central nervous system. The peritoneal cavity is especially liable to injury or secondary implication. In any case where a physician is called upon to examine the subject of a sexual assault it is his duty to proceed with rigid asepsis, to avoid the possibility of having secondary consequences attributed to his neglect.

Sexual congress with male children by adult females is in some of the states punished as indecent assault. The most serious consequences of such a crime are moral injury and venereal disease, but it is not an offense of great medicolegal importance.

Some of the questions that arise in connection with sexual abuse, short of intercourse, when practised on children call for discussion, because of the possibility of their raising a suspicion of the graver crime of rape. The disgusting possibilities of a less serious kind are manustupration practised on children; seduction of children to manustuprate the offender; seduction of children for sexual exhibition. But the most important cases of the kind are those in which female children are involved, and which raise the question of actual sexual contact. An appeal in such cases may be made to the anatomic evidence afforded by the genitals, remembering the frequency with which children manipulate their own genitals. Gentle manual irritation would leave no characteristic sign behind. The child confines her rubbing to the vestibule; the elder seducer is more likely to direct his manipulations to the vagina. The conditions found, if not absolutely indicative of sexual contact, as they can seldom be, must depend for interpretation upon less direct testimony.

UNNATURAL SEXUAL OFFENSES.

Incest.—Of the unnatural sexual crimes none is more offensive to general moral sensibility than the highest degree of incest. Legally, incest is intercourse of man and woman who are related in any of the degrees which law makes prohibitive of marriage.

Incest is unknown to common law, and is a statutory crime, punishable in many states as felony. The most aggravated form of the crime is that which combines incest with rape.

There is a universal repugnance in the human family to sexual relations between persons closely related by ties of blood. The moral sense of savages and civilized people is so offended by incest that it has given rise to the belief that incest is avoided in obedience to an innate instinct of aversion. Such an explanation of the fact is but a change in terms and no explanation at all. Attempts at explanation have been satisfied by the conclusion that the origin of this universal aversion to sexual congress with blood relations is the result of teaching made necessary by experience of the evil results to offspring of consanguineous marriages. Were this an adequate explanation, we should certainly find among savage people instances where the effects of experience had not yet led to avoidance of incestuous marriage—an observation yet to be made. A phenomenon of life, universal in its manifestation, must depend upon something more certain than accident of teaching derived from experience; the more since the experience of inbreeding is not under all circumstances or immediately disastrous to progeny. Observation shows that there is no inherent aversion to marriage of blood relations *per se*, but repugnance for marriage of those that have been associated intimately in the family, or, among primitive peoples, in the tribe. This aversion naturally gave rise to the once wide-spread custom of marriage by capture, the remains of which are still to be found in many matrimonial customs of civilized peoples.

The *raison d'être* of this aversion to incestuous alliances may be discovered by an appeal to known psycho-physiologic laws. It is only necessary to consider the psychology and physiology of sexual development to make this clear. Normally, the distinctive mental characteristics of sex are developed comparatively late; they may be delayed in their unfolding even for some time after the sexual glands have begun their functional activity. The primary period of childhood may be spoken of as practically asexual. This period of indifferent sexuality, physical and mental, normally extending over many years—fourteen to sixteen—is the period of development during which the deepest and most enduring impressions are made on the mind. These early impressions are

derived from associations with the family, relatives, or other individuals associated intimately by a common bond of union. In these surroundings the child, for years devoid of sexuality, has no understanding or capacity for sexual attraction; his attachments for mother, father, sisters, brothers, are all the result of the pleasure derived from the kindness and protection he receives. Living in this atmosphere, his feelings uncolored by sexual sensibility, the measure of his capability to respond to impressions made by his intimates is soon completely filled, and finally fixed long before his sexuality has become manifest in other than secondary sexual characteristics derived by imitation from those about him of his own sex. Thus, when puberty approaches, the child's capabilities of mental association and emotion related to the members of the family have been completely filled; there is, as it were, no room for further variations of sensibility in relation to them. Suddenly or gradually cognizance of a new emotion, new longings, arises as the sexual glands assume their functional activity. These new feelings, strange and ill-defined at first, cannot be referred to impressions derived from associates of the family—these have all been experienced many times, and give rise to emotions well known and fixed in nature; the new feelings must, if they arise spontaneously, be projected beyond known sources of experience, and ultimately be brought into association with mental impressions derived from individuals alien to the family circle. Should sexual emotion arise as the result of external impressions primarily, this, for the same reason, could be only as the result of impressions made by other individuals than those that have constituted the circle of childhood's intimacies.

The constancy of this extraneous projection of sexual emotion, when once directed, depends upon the psychologic fact that first impressions and associations, especially those of a sexual nature, are practically and almost universally determinate for life; once directed outside the family, sexual sensibility is never normally altered in its tendency.

This explanation of the normal relations of sexual feeling serves also to emphasize the characteristics that distinguish active sexual love from non-sexual affections. The love of husband and wife is active sexual love; intense as it may be and often is, it is easily destroyed, perhaps because it does not date back to childhood, and because it commonly depends upon satisfaction of both sensual and mental needs. Paternal and maternal love is a secondary development of active sexual love, less open to change than that from which it springs, because more selfish or self-centered in its nature. Filial love, the result of early kindness and protection, the very life of childhood, does not change if once developed, but lives on through separations and changes of association that come in obedience to the intense impulsion of sexual passion.

Absence of this aversion to incestuous relations may be due to a variety of causes. A child whose sexuality was prematurely developed as a result of individual peculiarity or evil teaching might be deficient in this respect until properly taught. If mental development were deficient or delayed, incestuous tendencies might naturally arise. The

normal instinct once acquired might be lost as a result of mental disease, or temporarily suppressed by preponderance of pathologic hyper-excitation of sexual desire. We should conclude, then, in any case of the higher degrees of incest, that there was a strong reason to investigate the mental condition of the culprit; but here, as in all cases where mental integrity is called in question, the proof of irresponsibility cannot be based upon an isolated act or mental symptom.

A minor sexual offense recognized by the law is **indecent exposure** or **sexual exhibition.** The law defines indecent exposure as such intentional exhibition of the naked human body or the private members as would shock the feeling of modesty or tend to corrupt morals. In common law the offense must be perpetrated in a public place and in the view of more than one person, but various statutes are of broader application.

Genital exposure is, in itself, where publicly committed, an act of most striking folly, marked by absence of all rational purpose. This fact makes the perpetrator of it a subject for mental investigation. Experience shows that exhibition is almost invariably dependent upon some mental anomaly, permanent or temporary. It may be done in intoxication, in satyriasis or nymphomania, in senile mental degeneration, or as an expression of sexual perversion.

The most disgusting forms of unnatural sexual offenses, while of comparatively infrequent occurrence, are yet medicolegally highly important; these include **pederasty, bestiality,** and the less important practice of **tribadism.** In English common law the term sodomy covers pederasty and bestiality, and is defined as carnal knowledge against the common order of nature, of man with man or woman, or of man or woman with a beast. Formerly, this unnatural crime was punishable with death; it is now punishable with ten years of penal servitude in England, but in the United States it is no more clearly defined than as an offense in common law. Penetration is essential to the legal definition of any form of sodomy. The evidence of an accomplice, if unsubstantiated, is not sufficient to prove guilt. Pederasty is legally defined as *immissio penis in anum.* Both parties to the act are, when voluntary and responsible participants, open to punishment. The offense may be committed by male with male or female. The male's part may be either active or passive; the female rôle is always passive.

The possibilities of this offense call for legal consideration similar to that required in the case of rape. One or the other or both of the parties to the act may, by reason of age or other disability, be legally irresponsible; the active party may accomplish his purpose by means of physical or moral force or fraud; the passive party may persuade an irresponsible active party to practise the vice.

Owing to the convincing evidence of physical signs, efforts have been made from the earliest times to establish certain conditions which are diagnostic of addiction to the vice. Tardieu thought that the practice of the vice made the glans penis pointed; constriction of the body of the penis at some distance from the glans was assumed to be the result

of constricting pressure exerted by the sphincter ani. Such peculiarities are clearly the results of developmental defect, and they suffice to show that there are no diagnostic marks of the active practice of pederasty. Abrasions of the penis might arise from forcible attempts to pass it through the sphincter ani, but such accidental marks would have no independent diagnostic value.

Passive pederasty is capable of giving rise to more unequivocal physical signs of its practice. Forced *immissio penis in anum*, especially in the case of children, may well cause abrasions and tears of the mucous membrane of the anus, or even more serious and extensive lacerations, and consequent inflammation; but severe injuries are much more likely to have been induced by preparatory use of other means to accomplish the necessary degree of dilatation. A relaxed sphincter with obliteration of the centrally radiating folds that are characteristic of the normal condition has been interpreted as evidence of the passive practice of the vice; but such conditions are not unequivocal evidence, for they may be due to a great variety of causes. The same may be said of hypertrophic growths about the anus and of proctitis. Venereal disease may, of course, be communicated by pederasty; but when the anus or rectum is the seat of such affections, it is to be remembered that they may have arisen in other common ways. Demonstration of spermatozoa in the person of the passive party would be conclusive in some cases; but rarely so, if the passive male party were beyond the age of puberty.

The degraded vice of bestiality cannot afford physical evidence of its practice. Animals made use of may show signs of the evil practice to which they have been subjected; demonstration of human spermatozoa in an animal would be conclusive proof. Almost any of the domestic animals may become a means of unnatural gratification.

The medicolegal importance of tribadism, or Lesbian love, is comparatively slight. In its fully developed form it consists of *immissio clitoridis in vaginam*, an act possible only when the organ is of unusual size and length—a condition that must be exceedingly rare. A female given to the active practice of the vice may have an enlarged clitoris, but such a peculiarity is no sign of such addiction. Those given to tribadism commonly resort to a minor vicious practice which might call for legal investigation—namely, genital kissing, or cunnilingus. This vice is practised as a part of tribadism, and is mainly of interest because it might be a form of genital abuse or seduction of children.

Reference has been made only to the physical aspect of the foregoing vicious habits; but their most important relation is that which they sustain to the mental state. Such unnatural offenses are in themselves sufficient to raise a question of the mental integrity of their perpetrators, even though, in themselves, they are not a demonstration of irresponsibility. It is a sad commentary on humanity that the impulses of passion may induce a degree of degradation and depravity equalled only by the depth of moral degeneration due to mental defect or disease. This aspect of the unnatural offenses is discussed in more detail under the heading of Perversions of the Sexual Instinct (Vol. I., page 683).

VENEREAL AND GENITO-URINARY DISEASES IN THEIR MEDICOLEGAL RELATIONS.

In treating of this branch of the subject it has seemed advisable to make the following divisions: Impotence and sterility as they exist in the male and female—their causes; curability or incurability; gonorrhea, its effects in men, women, and in children, and its bearing in legal medicine; syphilis, its effects in the same relations.

Impotence and Sterility.—As these terms are often used loosely, impotence being used where sterility is meant, and vice versa, it may be well at the outset to define them. By **impotence** is meant the inability to perform the sexual act, and by **sterility** is meant the impossibility of having children; thus a man may, from some physical defect, be incapable of copulation, although the seminal fluid may be secreted and contain all the elements necessary for fecundation, or he may have strong sexual desire and be perfectly capable of normal sexual intercourse and yet the element necessary for fecundation—*i. e.*, spermatozoa—be absent from the seminal fluid, or there may be no material ejaculated. Of course, both these defects may exist in the same individual and often do, and sometimes it may be difficult accurately to determine under which head a given condition should be placed.

Impotence in the male may arise from some physical defect in the genital organs, either congenital or acquired, the cause of which is obvious. There may be loss, deformity, or complete absence of the penis; some mechanical impediment to complete erection, due to defective circulation of the blood in the penis; arrest of development, or absence of the corpora cavernosa. There may also be some disease of the corpora cavernosa, such as syphilitic or gonorrheal infiltration, or the so-called fracture of the penis which occurs while in the erect condition. After healing has taken place, the portion of the penis in front of the point at which the fracture occurred, or the point of infiltration, remains flaccid on erection of the organ, or there may remain so marked a curvature of the organ that intromission is attended with great difficulty or is rendered impossible. Sometimes, however, complete recovery has followed this accident, the cicatricial portion has become absorbed, and the organ has resumed its normal condition.

A unique case was recently reported by Leflaive and Barbulee.[1] They were called to see a newly married man in whom attempted intercourse had been attended by great pain and swelling of the anterior portion of the penis. The patient gave a history of having, twelve

[1] *Bulletin médicale*, December 1, 1895.

years before, passed a brass ring over the penis, and the anterior portion of the organ had become so swollen that he could not remove it. He bore his suffering at the time, not venturing to tell any one, from shame. Meanwhile the ring ulcerated through the skin and the skin closed over it. It is astonishing that no further trouble arose until marriage. The ring was removed by incision and cutting with heavy forceps, and the case went on to complete recovery.

As an example of arrest of development the writer has under his care a man who has been married nearly a year, and not once during that time has he been able to obtain sufficient erection to have intercourse with his wife. He is twenty-two years old, the genital organs are rather small, and there is apparent lack of complete development of the corpora cavernosa, so that the erection is incomplete. He has a slight left varicocele. The man is of a mild disposition, and rather stupid. He states that at thirteen or fourteen years of age he began to have some sexual desire, and that erection, although never what it should be, was better then than now; he practised masturbation for several years; only twice did he remember to have had a nocturnal emission. Several times before marriage he attempted intercourse, but was unsuccessful; when he persists in attempts at intercourse there is finally an ejaculation. The examined seminal fluid was found to contain well-formed spermatozoa, but it was examined too long after ejaculation to determine whether they possessed active movement. The outlook for this case is rather a hopeless one, and, if it proves to be incurable, is, under the circumstances, a justification for the wife to obtain a divorce.

A short frenum has been known to be a cause of impotence, and phimosis also, but these defects are easily curable. Varicocele may cause impotence; this, too, in simple cases is curable.

Deformities, such as hypospadias and epispadias, when of marked degree, may interfere with the proper performance of the sexual act and be a cause either of impotence or of sterility. They are, however, more apt to be associated with sterility if the urethral orifice is situated so far back that the ejaculated fluid does not enter the vagina. When hypospadias is of marked degree, associated with adherence of the under surface of the penis with the scrotum, there is also impotence. These defects are often, however, capable of reparation by surgical operations and need not be considered further.

In contradistinction to the absence of or an abnormally small penis, an abnormally large penis, or a double penis, partial or complete, may give rise to impotence from inability to perform intromission, although cases have been reported in which a double penis existed, and in which copulation was successful and children were born.

Other forms of impotence associated with lack of power of erection, or evanescent erection associated with premature ejaculation, exist, the causes for which are various. Some of these seem to be dependent upon a nervous mental condition rather than a physical defect, but surgeons of the present day are apt to believe that the majority of these cases have some local disturbance as an underlying cause. It is notorious

that some men who before marriage have repeatedly indulged in sexual intercourse have found themselves, after marriage with a virtuous woman, temporarily, at least, impotent. Impotence may result sometimes through the nervous fear of failure; not infrequently because of a chronic hyperemia of the deep urethra, either from excessive sexual indulgence or from excessive masturbation or from chronic posterior urethritis the sequel of an old gonorrhea. It may also be found to be dependent upon some trouble at a distance, due to reflex nervous disturbance, such as a fissured anus, hemorrhoids, etc. Such cases, as a rule, are temporary only, and a little study, with the remedying of defects, will readily bring about a cure.

Impotence has been reputed to have followed in cases where internal urethrotomy has been performed, although in what manner this acts if there has been no apparent injury to the corpora cavernosa is not made clear, but these patients complain of inability to have an erection. It is, however, more than probable that there is also present a chronic prostatitis or seminal vesiculitis which has been overlooked. The writer has seen one such case in which inability to obtain an erection had existed for more than a year and was thought by the patient to have been caused by the operation, but marked improvement followed treatment directed toward the cure of a seminal vesiculitis and prostatitis which were found to be present.

Diabetes, chronic hypertrophy of prostate, and a large irreducible hernia may be mentioned as causes of impotence.

Sterility in the Male.—This may be due to several causes. It may be dependent upon impotence, the removal of the causes of which may be all that is necessary to a cure; it may be due to some condition by which the seminal fluid is unable to reach the vagina, although the semen itself is normal, as, for instance, a tight prepuce, and although the sexual act is capable of performance, the fluid is pocketed behind the narrow orifice. The existence of a tight stricture, whether from an old gonorrhea or traumatism, may prove an obstacle to the emission of seminal fluid, the portion of urethra behind the stricture being distended by the fluid, which may then gradually leak back into the bladder, to be voided in the urine. Generally speaking, these conditions are easily curable. Further, there may be a blocking-up of the orifice of the ejaculatory ducts from old gonorrheal disease, from prostatic calculi, old cicatrices, etc. Not infrequently cases have been reported of sterility in men consequent upon the performance of a perineal lithotomy in childhood, the ejaculatory ducts having been severed during the operation. Cases are also seen where there is at least a temporary impotence, or impotence combined with sterility, from perineal lithotomy in adults due either to extreme dilatation of the prostatic urethra or perhaps from injury to the ejaculatory ducts due to a large-sized stone. This condition may be due to extreme trauma or from the operation *per se*, for impotence, temporary at least, may sometimes follow simple operation on the genito-urinary canal.

Absence of testes, congenital or from castration, is obviously a cause

of sterility. It may be due to retained testes, where the testes remain in the abdominal cavity or in the inguinal canal. When one testis is in its normal position the man is generally not sterile, although he may be. Sterility may further be due to atrophy of the testes. Causes which lead to atrophy are varicocele when of long standing and of marked degree, traumatism, disease of the testes or epididymis, whether from syphilitic, gonorrheal, tuberculous, or malignant disease. Sterility, however, does not follow gonorrheal epididymitis so frequently as has been believed. In order to do so it must generally occur upon both sides, causing complete blocking of both vasa deferentia. The writer has not infrequently seen cases in which double epididymitis has occurred, and resolution has taken place upon one or both sides, the patients afterward being capable of procreation, although slight nodules may still be found present in the tail of the epididymis.

Dr. George T. Elliot related to the writer such a case: the patient, a young man who had suffered from double epididymitis, had repeated intercourse with a woman under the belief that impregnation was impossible. She afterward, finding herself pregnant, called him to account, and he, fully confident that an examination would prove him sterile, applied to Dr. Elliot, who, making a microscopic examination of his seminal fluid, found him rather uncommonly fruitful than otherwise.

The apparent power of spermatozoa to pass obstructions under certain conditions is well illustrated by a case reported by Dr. Bransford Lewis,[1] in which he ligated both vasa deferentia for an enlarged prostate. Microscopic examinations of material obtained by stripping the seminal vesicles, made at intervals for more than a month after the operation, showed the constant presence of spermatozoa in these organs. This led him to resect the ducts; and after this second operation, the spermatozoa were found to be present for about a week and then they disappeared, showing apparently, though not necessarily conclusively, that the spermatozoa, after the first operation, were able to pass the obstruction caused by the ligatures. This brings up rather an interesting question, whether shortly after castration a man may not still be capable of having children. Rosse[2] quotes 2 cases in which a husband presumably impregnated his wife after loss of testes—in one case loss was by a gunshot wound.

A more frequent cause for sterility and impotence than epididymitis may be found in acute or chronic inflammation of the seminal vesicles and prostate. Such an inflammation may be simple, gonorrheal, or tuberculous in its nature. It is most frequently gonorrheal. These inflammatory conditions may cause impotence in a manner already explained; there being inability to obtain a complete erection apparently due to this condition, or the erection may be evanescent, or ejaculation may be premature, occurring before intromission. They may give rise also to frequent pollutions and spermatorrhea. They may be a cause of sterility in one or more ways. There may be absence of ejaculation

[1] *Journal of Cutaneous and Genito-Urinary Diseases*, August, 1896.
[2] *Medical Jurisprudence*; Witthaus, *Forensic Medicine and Toxicology*.

from closure of the ejaculatory ducts, either from thickening of the mucous membrane of the walls of the ducts at the mouths, or the mucous membrane about the mouths, or cicatricial contractions; or there may be atrophy of the seminal vesicles; or weak muscular action of the walls of the seminal vesicles; thinning of the vesicular walls; partial or complete destruction from severe inflammatory conditions; a perivesicular inflammation binding down the vesicular walls—all these conditions may cause azoöspermia, or the spermatozoa may lose their vitality from the altered character of the secretions from the seminal vesicles and prostate, thus causing necrospermia. The secretion of the prostate seems to have for its purpose the preserving of the life of the spermatozoa, and when altered it may no longer possess this quality. When in this class of cases impotence only is present, the case is curable, but where the patients have gone on to sterility they may remain permanently and incurably sterile.

Azoöspermatism may also occur without discoverable cause. The man may be potent, but the seminal fluid is devoid of spermatozoa, or, if present, they are inactive. These cases are almost always incurable.

Impotence and Sterility in Women.—Impotence in women may be due to congenital defects, such as atresia or absence of vagina; a thick, tough, unyielding hymen, or malformations of the vagina, as where the vagina ends in the bladder or rectum. Narrowing of the pelvis from rachitic disease may be so great that intromission is impossible. These conditions may be partial or complete; some may be remedied by operation, some are incurable.

Another condition which might be called a true impotence is a condition known as vaginismus, a peculiar spasm of the muscles about the *ostium vaginæ*, occurring when any attempt at intercourse is made and preventing intromission, causing pain to the woman and horror on her part at any sexual approach of the husband. This condition may vary in extent and at different times, from a slight inconvenience to absolute impotence, a condition trying to treat and often incurable.

Sterility in the woman may arise from many causes, although sometimes the fault is wrongly placed upon the woman. In determining the cause of sterility both the husband and wife should be examined. There may be absence of the ovaries, either congenital or from removal by surgical operation; the uterus may also be absent; the ovaries and uterus may remain in a rudimentary state; there may be complete or partial occlusion of the uterine cavity, or occlusion at either extremity of the Fallopian tubes. Absence of the uterus is associated usually with absence of the vagina or with the malformation in which the vagina ends in the bladder or rectum. The writer remembers the case of a girl, about fourteen years old, who entered a children's hospital for operation for a lump in the groin. This was taken for an enlarged inguinal gland, but on removal proved to be a rudimentary uterus with both tubes and ovaries which had apparently come through the inguinal canal. It was undoubtedly a congenital condition, as there

was no trace of a hernia; the vagina, although small, was present, and rectal examination failed to reveal the uterus or ovaries in their normal situation.

Malposition of the uterus, such as marked flexion or version in either direction, may cause sterility, as also may an elongated cervix uteri with a small os. Secretions from the uterine canal of an altered consistence or condition may cause death of the spermatozoa and thus give rise to sterility. A simple catarrhal condition of the uterus has been known to cause sterility, the cure of which has been followed by impregnation. Gonorrhea and syphilis may be causes of sterility which will be considered more fully under their respective heads.

According to Rosse,[1] in English statistics 12 per cent. of marriages are sterile, and in 9 out of 10 cases the woman is at fault.

Cases may be seen where sterility exists and no apparent cause can be found present in either husband or wife, so far as modern methods of examination can determine. Some of these cases may be explained on the ground that they are sterile relatively to each other, although either might be found to be fertile with some other mate, and cases have been reported where both parties, on being separated and marrying others, have procreated children.

In English-speaking countries impotence or sterility as a cause for divorce must be shown to be incurable and to have existed before marriage.

Impotence or sterility arising from accident may give rise to suits for damages, and in such cases there would seem to be considerable room for malingering. I remember the case of a man who was struck by the pole of a truck and suffered an extensive laceration of the integument of the scrotum, exposing the testis on one side. The wound healed without trouble, but the man complained of complete impotence. He was a strong, powerful man, but it seemed to me that he was more interested in ascertaining the money value of his injuries than he was in ascertaining the means for recovery, and certainly exaggerated his symptoms. I soon lost sight of him, and so never knew the outcome.

Gonorrhea.—In considering the effects of this disease, I shall divide the subject into the etiology; its effects as found in men and in women; its effects upon their offspring; its existence in children, and some of the medicolegal aspects. It would take too much space to give all the reasons, array the cases, and quote all the authorities from which the writer has drawn the conclusions which he places here, and it has seemed best to state, as briefly as possible, what he believes to be the weight of authority.

This disease, which has come down to us from antiquity, is one of the most widely disseminated of the venereal diseases, and yet it seems as if we were only on the threshold of the knowledge which should be ours.

The manner in which gonorrhea is most frequently acquired renders the patient desirous of concealing its existence, and from a feeling of shame

[1] Witthaus, *Medical Jurisprudence.*

he applies not to his physician for advice, but to the corner drug-store. The profession, therefore, sees it comparatively seldom in its earliest stages, but more frequently after quacks, drug-clerks, and persons who from their own wide experience in acquiring the disease, consider themselves thoroughly capable of treating it in others, have expended their ingenuity upon it.

Only too long has the profession looked upon it as a disease of but slight significance, except in a small proportion of the cases, and to Noeggerath is due the first real warning note, which was sounded as late as 1872. Although, at the time, Noeggerath's statements were looked upon as greatly exaggerated, closer study seems to bear him out, and he does not appear to have greatly overstated the case. A large percentage of blindness from infancy, sterility in the husband or wife, or a life of invalidism on the part of one or the other, can be ascribed to its influence, and the disease contracted in the ignorance of youth, and even forgotten, has not infrequently been the cause of long-continued suffering on the part of the wife, marriage having taken place long after subsidence of the disease in the man.

Etiology.—The amount of careful study and close observation which have been made upon this disease since Neisser, in 1879, claimed to have discovered its true cause, in the germ described by him and named the gonococcus, has been vast. Gonorrhea, so far as we to-day understand it, in all its vagaries, in the vast majority of cases is due to a micro-organism known as the gonococcus. It is a bacterium possessing certain known properties. These are a definite shape; characteristic method of multiplication in pairs and fours; situation, as in the protoplasm of the pus-cells; reaction to certain mucous membranes; capability of growth on certain culture-media, under certain conditions. It may be obtained in pure culture after the growth of repeated generations, and when the isolated organisms are placed on the mucous membrane of the human urethra or conjunctiva, they are capable, provided they have not lost their virulence, of setting up, after a certain time, an acute purulent inflammation of that mucous membrane, which runs a definite course. The secretions, in turn, are found to contain the micro-organism in abundance.

There are numerous reports in literature which go to prove that the germ may be conveyed to the genital mucous membrane in an innocent and accidental manner, through carelessness or ignorance, and this, together with the fact that the mere placing of the germ upon such mucous membrane, without the occurrence of sexual intercourse, is capable of setting up the disease, forces us to the conclusion that the disease may be acquired in an entirely innocent manner.

Further, it is now a well-known and well-established fact that in a vastly smaller number of cases other micro-organisms are capable of setting up a urethritis or urethral inflammation, which to the eye and from the history of the case cannot be distinguished from a true gonorrhea, and a differentiation can be made only by the microscope and by culture experiments.

Most physicians adhere tenaciously to the opinion that gonorrhea can be acquired only by sexual intercourse, and, of course, in the vast majority of cases it is so acquired, in adults at least, but in their unwillingness to accept any other cause, through fear of being thought "soft" or of having too great faith in human nature, and because many patients do try to deceive, ascribing their plight to anything rather than the true cause, physicians may, at times, cause brutal injustice to innocent people. It is enough to know that such an inflammation, the discharge from which is found to contain gonococci, can have its origin only from a source in which there was a similar condition, but the manner of acquisition may have been innocent. The method may have been mediate contagion. A physician[1] having a large clinical and hospital experience in venereal diseases, while examining and experimenting with such cases, goes to the urinal and accidentally conveys gonorrheal pus to his own meatus, having neglected properly to cleanse his hands, finds himself a few days later afflicted with a gonorrhea, and reports his case only to expose himself to the ridiculing shafts of his colleagues.

Dr. F. Tilden Brown related an interesting case to the writer. A patient came to him suffering from a gonorrhea, and having nothing to conceal, disclosed to him its only possible source, a young woman with whom he had had sexual relations. She was examined and found to have the disease; both patients disclaim any other source. It was found that the sister of the young woman was under treatment for a gonorrhea and habitually used her sister's fountain douche and vaginal nozzle, making this then the possible, and indeed probable, source of the disease.

Cases are reported to prove that gonorrhea may arise *de novo*, without sexual intercourse, with the idea of refuting the specificity of the gonococcus. Such cases, to the writer's mind, tend to confirm, rather than weaken, the theory. Straus[2] reports the case of a boy, sixteen years old, who had never had sexual intercourse, but had practised masturbation to excess. He came under observation with an acute purulent discharge containing numerous gonococci, a few days after an excess of this nature, the case going on to suffer from a typical gonorrhea. To the writer's mind it is far easier to conceive how gonorrheal pus could reach the boy's urethra at such a time than it is to conceive how the disease could arise *de novo*. If the latter were the case, we would certainly see many more such cases.

One of the most persistent statements of writers, even in the most recent text-books, who reluctantly accept the rôle which the gonococcus plays in this disease, thus following Ricord, whose observations were made before the discovery of the gonococcus, is that a woman who has never had a gonorrhea and in whom no trace of it can be found, is capable of giving gonorrhea to men—*i. e.*, the man, they say, is the cause of his own gonorrhea. The answer to this statement, it seems to me, may be better made in the paragraph on the Effects of Gonor-

[1] *Monatshefte fur praktische Dermatologie*, July, 1896, p. 16.
[2] *Archives de médecine expérimentelle et d'anatomie pathologique*, 1889, 1, p. 326.

rhea in Women, although the proof of the existence of latent gonorrhea will thoroughly explain these cases.

Latent Gonorrhea.—By latent gonorrhea is meant, briefly, a condition in which gonorrhea has occurred and quieted down; patients in whom there may or may not be a discoverable lesion, but in whom the most thorough search by the microscope will not reveal the presence of gonococci, and yet these patients may be at one time or another a source of danger to others. At the present time it is known that gonococci are capable of assuming what is known as an *involuted* form.[1] Gordon[2] refers to this, saying: "Wertheim only recently was enabled to give a thoroughly scientific explanation of latent gonorrhea. He says that only young gonococci are recognizable, as they are stained by anilin solutions, while old gonococci lose their typical forms by becoming granular spheres, variable in size and indefinite in outline. This change occurs whenever the culture-medium is exhausted and no longer nutritious. He proves this by transplanting the afore-mentioned altered forms into fresh culture-media and raising typical gonococci. The logical consequence is that patients proclaimed cured when the microscope revealed no gonococci, may possess them in their latent form, which can be detected only when transferred to a better soil or culture-medium, when typical gonococci are raised."

Effects of Gonorrhea in Man.—The interest in this centers mostly upon the possibility of conveying the disease to innocent people. First to be considered is the wife whom he is to marry; secondarily, the effects of the disease upon himself.

The disease is more widely spread among the male sex, and wives are more frequently infected by their husbands than *vice versâ*. It is difficult to say in which sex the disease may produce the most suffering. A woman may acquire the disease and not know that she has it, but it is probably very seldom that a man can acquire it and be ignorant of the fact, although this is within the bounds of possibility.

A man may have a gonorrhea, go through a typical attack, and never hear from it again, and thousands of such cases marry and never infect their wives: some, because they have marked powers to resist disease, others because they take care of themselves, receive skilful treatment, and avoid excesses and exposure to a second attack. Some are careless and have repeated attacks; their habits are not of the best, and they have not enough self-restraint to go through the tedious treatment necessary to produce a cure; they tire of treatment, suffer relapse, and try again, and believe there is no cure. These cases are probably a majority of those that are a source of infection. Briefly, the sequelæ in men are granular patches of unhealed ulcerations, minute in size perhaps, in any portion of the urethra; chronic infection of the glands and crypts of the urethra; stricture formation; chronic anterior and posterior urethritis; chronic prostatitis, seminal vesiculitis, and perivesiculitis, and epididymitis. These latter have already been mentioned in

[1] Wertheim, *Centralblatt für Gynäkologie*, 1895, No. 45.
[2] *Medical Record*, 1896, No. 5, p. 740.

speaking of sterility and impotence. The effects upon the bladder, ureters, and kidneys are hardly necessary to mention here. Acute gonorrhea and its complications and their effects in producing infection in others need not be mentioned, as they are easily understood.

The important thing from a medicolegal standpoint is the length of life of the gonococcus, its capability of undergoing involution and remaining in a latent form, difficult to recognize, and yet capable, when transferred to a fresh mucous membrane, of springing into life and producing the disease in others. Men who have had the disease believe themselves cured when it no longer gives trouble, and when there is no external visible sign. Some, from knowledge or conscientiousness, consult the physician as to whether it is safe for them to marry. Kopp[1] relates the cases of 6 men whom he had allowed to marry after having made repeated examinations of the shreds and "Tripper-faden" in their urine and not finding gonococci present. All 6 shortly after marriage infected their wives, producing in them a typical gonorrhea containing the gonococcus. The writer has repeatedly seen men whose urine for long periods of time was absolutely clear to the eye, and yet material of a pathologic nature of gonorrheal origin has been obtained from the prostate and seminal vesicles, stricture and lesion of the urethra being absent. It must also be remembered that a gonorrhea may pass over a urethral mucous membrane and leave it in such a condition that other micro-organisms gain a foothold and set up chronic troubles, while the gonococcus may have entirely disappeared, and yet some of these cases are capable of setting up a condition of unsound health in the wife.

The question is, Can a physician safely advise such a patient to marry? Thousands of such cases do marry and no evidences of trouble appear in their wives, but in thousands of such cases trouble does arise. It would seem, therefore, that the physician has gone to the extent of his duty when he has put the patient in the best possible physical condition, but he would be wiser not to take the responsibility of advising marriage.

Effects in Women.—One of the peculiar effects of gonorrhea in married women is that husband and wife may have sexual relations without giving rise to fresh attacks in either—that is, they have become "acclimated"—and yet should another man have sexual relations with the wife, he may easily acquire the disease. As has been said, women may have gonorrhea and never know it—that is, it does not give them enough discomfort to consult a physician, and they are unsuspicious, and when, at a future time, the question comes up whether they have ever had the disease, they may, with belief, deny that they have had it, and in a superficial examination or even a careful one they may not show any trace. The disease has assumed a latent form. On the other hand, the husband, either from overindulgence in sexual intercourse or from intercourse near the menstrual epoch, or from some unknown cause, may have a fresh outbreak of the disease and suspect the wife of infidelity. The manner in which this is supposed to occur is explained by

[1] *International Surgery*, November, 1898, p. 307, editorial on Kopp's paper.

Wertheim.[1] He made cultures of the gonococcus from the urethra of a man in whom the disease was quiescent. He cultivated the germ for several generations until the medium was free from all foreign substances. Although he made repeated attempts, he failed to reinoculate the man's urethra, and yet, from this same culture, inoculation of another urethra produced a typical gonorrhea. From the secretions of this infected urethra he again made cultures, and from these new cultures he was able to produce a typical gonorrhea in the original urethra which had proved to be immune to its own cultivated gonococci. From this Wertheim concludes that the man does not awaken an old gonorrhea in himself, but is reinfected from the gonorrhea which he has produced in the wife.

Gordon[2] says: "Gonorrhea in women manifests itself in one or more of the following affections: Vulvitis, bartholinitis, urethritis, vaginitis, metritis, parametritis, salpingitis, oöphoritis, and peritonitis. Sometimes the disease extends from the urethra, causing gonorrheal cystitis, ureteritis, and nephritis," and, of course, the metastatic forms, as arthritis, endocarditis, etc., are met with as in men or in children.

The possible effects of the disease are abortions or sterility. A few of the causes of sterility from this disease are altered secretion from the uterus and genital tract, in which the spermatozoa lose their vitality; alterations of the mucous membrane of the uterus, so that the ova fail to get a start; stenoses of the uterine canal, which offer obstacles to entrance of spermatozoa, or stenoses or altered secretions of the Fallopian tubes, which cause death of the ovum, etc.

Further, these various troubles may entail life-long suffering; may prove dangerous to life; in childbirth there may be an autoinfection, causing a form of puerperal fever. On the other hand, women may have gonorrhea and never suffer any discoverable inconvenience from it.

Effects of Gonorrhea upon the Offspring and in Children.—The most frequent effect produced is gonorrheal ophthalmia, which appears a few days after birth. These infants may also have a gonorrheal inflammation of almost any other portion of the body, such as arthritis, pleuritis, meningomyelitis, etc.

Gonorrhea in one member of a household may spread to other members in an almost unaccountable manner. Thus, a few days after birth the new-born infant may have gonorrheal ophthalmia, and a few days later an older child may develop a gonorrheal vulvovaginitis. In institutions where children are collected it has come to be only too well known that if a child with vulvovaginitis is introduced into the institution, the disease may become epidemic, and few of the female children escape. It is only in recent years that intelligent study of these epidemics has been made. Many of them are found to be due to a true gonorrhea, and the ways are devious by which it spreads; the baths, towels, sponges, even thermometers, where the practice is to take the temperature by the rectum, the bed-linen, etc., have been shown to be the agents by which

[1] *Wiener klinische Wochenschrift*, 1894, No. 14.
[2] *Medical Record*, November 21, 1896.

the disease has spread. It is to be noted that this disease, as it appears in such institutions, spreads more rapidly among the girls, but in a recent outbreak in an institution in New York,[1] though the spread was rapid among the girls, one boy developed an acute ophthalmia from using a towel used by a girl who had a vulvovaginitis, and another boy, six years old, acquired an acute urethritis, it was thought, from using a common chamber. It may be said in passing that other infectious forms of vulvovaginitis in young girls exist, but I am speaking only of gonorrhea.

It would seem that the anatomic form of the genital tract renders girls more susceptible to this trouble than boys. Certainly it is seen far more commonly in girls, and the question may be asked, at this point, whether some of the unaccountable cases, as where gonorrhea has apparently been contracted by a man from a young woman who does not show any trace of the disease and never had sexual relations with any other man, may not be explained by the fact that in early life she had such a vulvovaginitis.[2]

The effects of vulvovaginitis in young girls may be slight or extreme. Many possible complications may arise. It may cause a peritonitis, a gonorrheal source being unsuspected, or it may endanger life.

A mother finding her child with such a disease might easily suspect that the child had been tampered with and endeavor to throw the blame on an innocent person, but the disease appearing in a very young girl without marks of violence, would far more likely be due to an accidental infection than to one of criminal assault. In fact, the cases in which it is accidentally acquired far outnumber the instances by which it has been acquired from evil intent. Walker,[3] in examinations of 12 cases for supposed rape, found the disease only once. Spaeth,[4] in 21 cases of vulvovaginitis in children, found that 14 were gonorrheal, and of these 14 patients, 11 of the mothers were found to be suffering from chronic gonorrhea.

Martin[5] found a gonorrheal vulvovaginitis in a child five months old who had never been out of the mother's sight. The mother had had leukorrhea of long standing, but gonorrheal disease was not discoverable. Secretions from the infant's genitals inoculated upon the urethra of a man produced a typical gonorrhea.

As before mentioned, gonorrhea is not so common in boys as in girls. The effects in boys may be much the same as some of the effects in men. Abbe reported a case of gonorrhea in a boy of three years, followed by stricture of the urethra which necessitated operation. The disease was apparently contracted from the nursemaid. Koplik[6] reports 3 cases of gonorrhea in young boys. Two of these, aged five and nine

[1] Sheffield, *American Medico-Surgical Bulletin*, 1896, p. 726.

[2] An idea of the prevalence of this trouble in infants and young girls may be obtained from papers by Martin, *Journal of Cutaneous and Genito-Urinary Diseases*, November, 1892; Koplik, *ibid.*, 1893, pp. 219 and 263; Sheffield, *American Medico-Surgical Bulletin*, 1895, p. 747

[3] Martin's paper, *loc. cit.*

[4] *Munchener medicinische Wochenschrift*, 1889, vol. xxxvi., p. 371.

[5] *Loc. cit.*

[6] *Loc. cit.*

years, were contracted from a young girl by attempted intercourse, and one was contracted from another boy. The writer has seen several cases of gonorrhea in boys ranging from eighteen months to ten years and older; in the cases under six years it has not been possible to determine the source.

Important as it is to make thorough search, in a suspected case, for the gonococcus in order to establish the fact that the organism found present, by microscopic examinations and by culture experiments, corresponds in every respect with the known properties of the gonococcus of Neisser, still even then, in medicolegal cases, the time has not come when it can be used alone as an absolute test, but it may be introduced as one fact in a chain of evidence. A. Haberda[1] has made some important experiments bearing on legal medicine. He allowed pus in different thicknesses of drops to dry upon small pieces of linen, and sought to establish—(1) Whether by microscopic examination the gonococci could be recognized by means of its form, size, position, and peculiarity to coloring-agents in these dried spots, and for how long a time it would be possible to recognize them. (2) Whether from such dry spots they could be cultivated by the Wertheim method. (3) Whether such pus could cause gonorrhea. He discovered that—(1) In thick drops it was possible to recognize the gonococcus after eight months; in thinner layers there was a limit of from days to weeks. (2) Culture-media remained sterile after the secretions had thoroughly dried. (3) The gonococcus seems to lose its virulence and to be incapable of producing a gonorrhea after thorough drying of the secretions.

Syphilis.—The new era of medicine has added as yet but little to our knowledge of the effects or of the treatment of this disease, but the same close, careful, painstaking observations have gone on here as well as in other departments of medicine. That it is a disease of microbic origin is generally believed, but the agent has as yet eluded all efforts to recognize it. Some old theories have been overthrown; new light has been let in here and there, but it can hardly be claimed that one forward step has been taken in the last ten years.

That the disease is one of the most far-reaching in the social fabric is not to be gainsaid, for, aside from the fact that although in the majority of instances it is acquired by sexual contact with one suffering from the active manifestations of the disease, it may be acquired in the most innocent manner, the length of time which elapses between the time that the disease is implanted and the time that it first manifests itself in the form of the chancre or initial lesion, frequently makes it difficult to trace the source. Further, the chancre may be so situated, or be of so slight a character, that it may be overlooked, and the disease may not be recognized until the so-called secondary manifestations appear. Thus a much longer period may pass by, making it still more difficult to trace the source. The public still needs education on many points in order to avoid needless exposure to contagion, and the most important duty on the part of the physician is faithfully to drill his

[1] *Centralblatt für Gynäkologie*, 1895, p. 145.

infected patient on the means he should employ to lessen the danger of giving the disease to others. Such persons, unfortunately, are very apt to be careless and forgetful, even though they would not knowingly convey the disease to others. A young man having syphilis, while in company, playfully applied the lighted end of his cigar to the lips of a young woman; filled with remorse at his brutal horseplay, he tenderly kisses her on the burned spot, and she later develops a chancre at that place. A sailor, with mucous patches in his mouth, tattooes a number of young men, moistening the tattooing needles by passing them over his tongue, and they each later develop a chancre at the site of the tattooing. A lady, while in her kitchen, tastes from the same spoon as does her syphilitic cook and develops a chancre of the lip. Barbers' razors and clippers, dental instruments, surgeons' instruments, towels, pipes, and numerous other articles have all been the means of conveying syphilis to innocent persons. Healthy children, from being promiscuously kissed by relatives or strangers, have been found to acquire syphilis from a known or an unknown source.

The most important questions in syphilis are in relation to marriage and its effects upon the offspring. In considering these questions it is well to consider its effects in men, in women, and upon their offspring.

In considering the effects of syphilis in a man the main interest centers about two questions: Can he infect his wife? Will his children show the effects of the disease? While he shows active manifestations of the disease there can be no question of the danger of infecting the wife. The most active sources of contagion are secretions from the initial lesion and from mucous patches in the mouth, the blood, and the secretions from the skin-lesions. As time passes these lesions may disappear; still the blood remains a possible source of danger. The disease may slumber, show no signs, then break out again. In a general way, the older the disease, the less effect it has upon the bearer, and the more thorough the treatment, the less danger there is that the wife will be infected. Later, long after symptoms have ceased, he may be the bearer of another class of lesions. These, generally speaking, are no longer contagious, although some cases have been reported, which appear to be open to doubt, especially where late buccal lesions have occurred by which a wife has apparently been infected. In reports of this kind it is absolutely necessary to subject them to the closest scrutiny before accepting the probability of contagion from such a source, for it is far easier to conceive the possibility of the wife being exposed to a more recent syphilis, whether innocent or illicit, than it is to conceive such a lesion to be contagious. Dr. Otis's[1] famous case, which need not be written out, shows how easy it is to accept the apparent, and what difficulties surround the getting at the true, cause.

The second question involves the possibility of inheritance of the disease by the child from the father through the seminal fluid, without the mother being affected: First, sterility in the father may be due to syphilis; this almost always is amenable to treatment, and need not be

[1] *Clinical Lectures on Syphilis and the Genito-urinary Diseases*, 1888, p. 47.

considered. Second, there seems to be no question that paternal syphilis may affect the offspring without the mother being infected, and that, too, long after the time when it has ceased to show active signs in the father. The physiologic secretions, if not mixed with secretions coming from pathologic lesions, are supposed to be innocuous, and attempts to inoculate syphilis from the seminal fluid have failed; still this is no proof that, along with the impregnating spermatozoa, the syphilitic germ may not be implanted with it. That non-contagious syphilitic manifestations may be conveyed to the child from the father is easy to conceive, but when a child is actively syphilitic, capable of infecting others, then accidental infection of the child from a fresher source must be absolutely eliminated before this can be charged to the father's account.

Syphilis in Women.—Like the husband, a woman may acquire syphilis before marriage; she may be infected by the husband before conception, or she may be infected by the husband after conception. If infected before marriage or by the husband before conception, the results are easily understood and accounted for. In general, syphilis, in its effects upon the woman, does not seem to be so severe as in man, but the effects on the offspring are more pronounced. Women may remain sterile or abort repeatedly; they may have still-born children or syphilitic children as a result of infection. Treatment and time have an attenuating effect. At the same time that these phenomena occur women may not show active effects of syphilis in their person, and this is one of the curious phenomena of the disease.

A woman may bear a syphilitic child; she may suckle it and fondle it, and yet at no time show any syphilitic manifestations, and still the child may be a source of danger to others. This is known as Colles's law. The explanations are: (1) That she has a latent syphilis (Fournier). (2) That she is immunized, by absorption of serum, toxins, and antitoxins, through the placenta (Finger). (3) Inasmuch as she is capable of having tertiary manifestations later, there is absorption of toxins through the placental circulation, which places her in the position of one who has gone through the secondary stage, and leaves her at the tertiary stage; that is, she has a latent tertiary syphilis (von Dühring).

Further, a woman may give birth to a syphilitic child, and, by suckling it, receive infection by having a chancre of the nipple, an exception to Colles's law. Such cases have been observed by Guibout, Rouse, Scarenzie, Lucas, and many others. At the same time, in investigating such cases, a fresh, acquired syphilis on the part of the child must be eliminated. Further (Morrow), women have borne syphilitic children from a syphilitic father, and healthy children from a healthy father, without showing signs of the disease.

Effects in Children.—Absolutely healthy children have been born of actively syphilitic parents, and have never shown the effects of the disease, and children have been born healthy, and years after have shown tertiary manifestations, or they have been born healthy and shortly after birth shown syphilitic disease. Children born of a syph-

ilitic mother have remained healthy although she suckled them while she was actively syphilitic, and still have remained healthy, or late in life (eighteen to twenty years) have manifested tertiary lesions. This is known as Bofetas's law; it is the reverse of Colles's, and is explainable by immunization through the placental circulation.

Children may be born having active syphilis, and be capable of infecting others, or they may have lesions which, to the eye, are the same, and yet be incapable of infecting others. The cases where a healthy nurse has been infected by a syphilitic child through suckling it are rare,[1] yet entirely possible, and are to be feared when a syphilitic child is to be nourished by a wet-nurse. The question on this very point may arise, whether the child has infected the woman or the woman has given syphilis to the child. The following may be the case: (1) The nurse may be actively syphilitic and infect the child, and yet show no active signs. (2) She may have a chancre of the nipple derived from a previous nursing. (3) She may be infected by a syphilitic child. (4) The child may get a chancre of the lip from being kissed by a syphilitic person, and infect the nurse by causing chancre of the nipple. (5) The nurse may get the chancre of the nipple from an outside source.

Syphilis has recently been adjudged a ground for divorce in France. If innocently acquired, it is difficult to see where there is any more justice in granting divorce for syphilis than for tuberculosis when it has occurred after marriage. If acquired from sexual contact, unless the partner in marriage also has it, it is presumptive of adultery, and therefore, ground for divorce.

[1] Barker, *Edinburgh Medical Journal*, June, 1896, p. 1124.

MARRIAGE AND DIVORCE.

BEFORE discussing the medicolegal topics that are usually comprised under this head it is necessary to review briefly the marriage institution from a purely legal standpoint.

Marriage at its inception is a contract: it can be entered upon like any other contract, and is, to a certain degree, subject, like other contracts, to indispensable and prerequisite conditions as to parties, consideration, subject-matter, and assent. It may be said in general that immediately upon the consummation of this contract, in which the state is deemed to be a silent participant, there is created among these three—namely, the two spouses and organized society—a status of mutual rights and duties. This is to some extent true even as to those marriage contracts which can be avoided legally. But the law endeavors to strengthen a valid marriage, and even tends to validate, under certain conditions, some marriages that might become voidable; and it is not only by the statute, but also by judicial precedent, that "the law favors marriage." As a consequence of this peculiar triangular relation, marriage, whether considered as a contract or as a status, differs materially in its legal incidents and effects from ordinary contracts. Thus, the common grounds for voiding and rescinding other contracts, such as duress, fraud, and incompetency, have frequently less force and are more readily deemed waived in the case of marriage; and even for certain gross frauds effected by false antenuptial representations as to health, temperament, social position, or worldly estate, the law affords no relief whatever.

Among elements or consequences of this status may be noted the right to dower and courtesy; the incident of privileged marital communications; the doctrine of the wife's right of support and the corresponding duty of the husband to supply her with necessities; the voiding of deeds, wills, and contracts that would operate in restraint of marriage; and the peculiarities of divorce and procedure. Broadly stated, the object of the state in interfering with the marital relation when once established is the protection of the family and the preservation and improvement of the species. The state does not, however, as a rule, attempt to regulate the relation before it arises. The general, almost universal, absence of such regulation has been a fruitful theme of discussion. That the breeding of the human race should be, as it is in most instances, so fortuitous and anarchic, is at first glance shocking to the reason; but upon further examination the difficulty of effecting any change from the present system—or no system—will become readily apparent. And yet, it is to be hoped that the legal authorities

may, at a not distant date, call upon the medical profession for the means of gradually undertaking the duty of preventing or regulating improper marriages among the dependent, delinquent, and deficient classes.[1] At present medicolegal questions concerning marriage arise not from the preventive, but from the remedial, law as to marriage. As a rule, therefore, it is in cases of annulment, divorce, and separation that the advice and testimony of the physician as to marital relations are sought by the attorney or judge.

Bishop defines marriage as "The civil status of one man and one woman united in law for life for the discharge to each other and the community of the duties legally incumbent on those whose association is founded on the distinction of sex." Stewart says that "A legal marriage is the union of a man and woman in the lawful relation of husband and wife whereby they can cohabit and rear legitimate children." According to an earlier definition, "Marriage is a contract having its origin in the law of nature antecedent to all civil institutions, but adopted by political society and charged thereby with various civil obligations. It is founded on mutual consent, which is the essence of all contracts, and is entered into by two persons of different sexes, with the view to their mutual comfort and support, and for the procreation of children."

Not only do great international differences in the law of marriage exist, but in the United States each state is, as to marriage legislation, independent and sovereign, the jurisdiction of the Federal Government as to marriage extending only over the District of Columbia, the territories, and our consulates. The conflict of law as to marriage and divorce has reached an almost scandalous notoriety. There is, perhaps, only one general rule that can appropriately be noticed here; this is that a marriage, if valid where it is made, is also valid and will be recognized as such everywhere else. No such difference exists in any other class of legislation as that regarding divorce, the grounds of which, if strictly proved, may be a cause only for a separation—*i. e.*, limited divorce—in one state or country, while in another jurisdiction exactly the same grounds may be liberally admitted, and, on slight evidence, as reason for total divorce. In some states a ground for avoiding the marriage *ab initio* is given under the causes for divorce, and annulment is also looked on as a species of divorce. There are, perhaps, no two states that agree as to what facts of residence, etc., will give jurisdiction in divorce proceedings. In some the distinction between a void and a voidable marriage may be discovered rather by reference to precedent than to statute; and there is a real necessity that

[1] Legislative effort in this direction is steadily assuming more definite shape. Both the Ohio and Minnesota State Legislatures have recently passed restrictive measures. In the State of New York Dr. Rulison, of Buffalo, is authority for and proposes the enactment of a statute dividing the community into three classes: Class A, to include only such persons as can furnish proof of having untainted physical and mental ancestry for at least three generations; Class B, including those persons where the record is clear for only two generations; Class C, including all other persons. Intermarriage between the various classes is to be prohibited.

all these distinctions be recognized, for upon them may depend such important matters as the legitimacy of children and the right of dower.

To illustrate the difference between void and voidable marriage let it be supposed that, in a given state, a bigamous "marriage" is *ipso facto* void *ab initio* without decree to that effect, and has been so determined, perhaps, in a purely collateral proceeding. The issue of such bigamous relation will, in the absence of statute providing for such a case, be illegitimate, and neither dower nor courtesy will arise from it. But if one of the parties to a marriage enter into the relation incurably insane, such marriage is, nevertheless, generally valid until declared void by a competent court; and unless it is so declared, all the incidents of marriage and the legitimacy of children will arise the same as in a perfectly valid marriage. This is often the case in states where certain marriages are forbidden as "void." But if a decree totally avoiding the marriage is obtained, the result is, in general, the same as if the marriage were void *ab initio*. In the absence of statutes protecting innocent offspring of such marriage, the latter will be illegitimate. No two states agree as to what marriages are void and what are voidable, but it may be said in general that the innocent offspring of such marriages are made legitimate by statute.

The New York statute, among others, may be referred to as affording a logical classification of matrimonial actions. It provides for three forms of such suits: first, an action to annul a void or voidable marriage; second, an action for divorce; third, an action for separation. All the grounds for such suits are set forth in the following sections from the New York statutes.

1742. Action by a Woman, Married under Sixteen, to annul Marriage.

An action may be maintained by the woman to procure a judgment declaring a marriage contract void, and to annul the marriage under the following circumstances:

1. Where the plaintiff had not attained the age of sixteen years at the time of marriage.

2. Where the marriage took place without the consent of the father, mother, guardian, or other person in legal charge.

3. Where it was not followed by consummation or cohabitation, and was not ratified by any mutual assent of parties after the plaintiff has attained the age of sixteen.

1743. Action may also be maintained to procure judgment declaring the marriage contract void, and to annul the marriage for either of the following causes existing at the time of the marriage:

1. One or both of the parties had not attained the age of legal consent.

2. That the former husband or wife of one party was living, and that the marriage with the former husband or wife was then in force.

3. That one of the parties was an idiot or lunatic.

4. That the consent of one of the parties was obtained by force, duress, or fraud.

5. That one of the parties was physically incapable of entering into the marriage contract.

But an action can be maintained under this subdivision *only* where the *incapacity* continues and is incurable.

1752. An action to annul marriage on the ground that one of the parties was physically incapable of entering into the marriage state may be maintained by the injured party against the party whose incapacity is alleged; or such an action

may be maintained by the party who is incapable against the other party provided the incapable party was unaware of the incapacity at the time of marriage, or, if aware of such incapacity, did not know it was incurable. Such action must be commenced before five years have expired since the marriage.

In an action for divorce the law provides but one ground—adultery.

A legal separation may be decreed for the following causes: 1. The cruel and inhuman treatment of the plaintiff by the defendant. 2. Such conduct on part of defendant to plaintiff as may render it unsafe and improper for the former to cohabit with the latter. 3. Abandonment of the plaintiff by the defendant. 4. Where the wife is plaintiff, the neglect or refusal of the defendant to provide.

The grounds for all sorts of matrimonial actions throughout the states are adultery, impotence, mental incapacity at time of marriage, mental incapacity during marriage, bigamy, abandonment, disappearance, cruelty, habitual drunkenness, antenuptial unchastity, lack of support, imprisonment for crime, conviction of felony, concealed antenuptial pregnancy not caused by husband, lack of age and consent, and loathsome disease. In some states practically any one of these grounds is sufficient for a total severing of the marital relation, either by a total divorce or by a nullity suit. In others, only a separation *a mensa et thoro* is granted for the less grave of the causes enumerated. In some of the states certain of the above grounds are legal basis for any suit, and each state differs from the others as to the causes for the different classes of action as well as to the jurisdictional facts, such as residence. In South Carolina a divorce cannot be obtained for any cause.

The states differ also as to the defenses to these actions. Some of these defenses are recrimination (counter-accusations), condonation (conditional forgiveness), collusion, connivance, and lack of volition. Cruelty in some states is allowed as a recrimination to adultery. The same evidence that is used to prove adultery is sometimes applicable and is used to prove illegitimacy. Such circumstantial evidence of inclination and opportunity which forces the irresistible conviction on the mind that adultery has been committed is admissible and sufficient proof of the latter. In general, the weight of evidence as in the civil cases is sufficient. As the state is interested in sustaining the marriage, the plaintiff's case must be proved in default cases, and in some jurisdictions a public officer acts in the state's interest on behalf of the absent defendant.

The only sexual crimes that are necessarily against marriage are adultery and bigamy. Incest, rape, seduction, and unnatural crimes are sexual crimes that may or may not be involved in offenses against the marriage relation.

Adultery is the voluntary sexual intercourse of a married person with one not the husband or wife. The definition of adultery considered as a crime is by no means uniform throughout the states. In some states, as in New York, adultery does not exist as a crime.

Bigamy is committed by any person who, being married, shall marry any other person during the life of the former husband or wife, unless legally divorced therefrom.

Cruelty is such conduct in one of the married parties as renders

cohabitation physically unsafe to a degree justifying a withdrawal therefrom.

On account of the technicalities connected with marital suits, and of the variance, already referred to, in the law of different jurisdictions, the above sketch of the more purely legal side of the subject of marriage and divorce is necessarily crude.

MEDICOLEGAL QUESTIONS.

The principal issues arising under the head of marriage and divorce in which law calls upon medicine are those involving questions of mental and physical capacity, cruelty, venereal disease, pregnancy, and the existence of sexual intercourse.

"Impotence," Physical Incapacity.—This may be a ground either for separation, divorce, or annulment, according to the jurisdiction. It must date from the inception of the marital relation. It is apparent that this is peculiarly a matter of medical evidence.

While the lack of physical capacity to consummate marriage is sometimes called cause for divorce, this term is really a misnomer, for decrees based on this ground are really decrees of nullification.

Strictly speaking, impotence means the want of procreative power in one of the contracting parties, whether arising from a faulty condition of the external or internal organs of generation, or from any moral or physical cause.[1] But from a legal standpoint, as an impediment to marriage or as a cause for its annulment, the defect must be relative to the capability of proper copulation, rather than mere inability to procreate. While it is often an undeserved misfortune to the innocent party in a suit on this ground that the physical side of the question should be aired in court, it must not be forgotten that it may be a moral as well as a physical necessity that each party should be provided with a remedy by the law. This becomes evident upon a short reflection on the habits and exigencies of spouses living under the conditions ordinarily possible. And in this connection it should be noted that the court (who is frequently a referee or master) can order the court-room cleared in the interest of public morality.

At the outset, some of the presumptions in these cases may be noticed. The power to consummate the marriage is presumed. The refusal of the copula from the date of marriage is *prima facie* evidence of impotence; yet in some jurisdictions is not evidence of desertion. Again, the physical consummation of marriage is more strongly presumed when there has been access of the husband; sleeping in the same bed or in the same room is stronger evidence, raising stronger presumption, than merely sleeping in the same house or apartment. In any case of alleged impotence the refusal to accept reasonable and proper means of cure is a conclusive presumption. Very delicate questions arise as to what means may be refused. What kind of impotence is it for which the law affords relief? It is not the failure to ejaculate nor the failure

[1] Wharton and Stille, *Medical Jurisprudence*.

to fecundate either the party to the suit or in general; nor the failure to be fecundated by the party in suit or in general, but the impossibility of the proper performance of the act of copulation between the parties to the suit. The power of either of the spouses to have copula with another than his or her marital partner is a totally irrelevant consideration.

It is not *impotentia seminalis*, but *impotentia copulandi* only in the individual case that constitutes legal impotence.[1] The law seems in this instance to take no note of the propagation of the race as one of the objects of matrimony, but rather of the gratification of desire.[2] The difficulty of proving that copulation could take place without fecundation has much to do with this result, and so the rule is subject to certain exceptions. In the event of castration or complete bilateral ovariotomy it is possible that the impossibility of fecundation would become relevant; and that the patent impossibility of parentage, one of the chief objects of marriage, would be taken into account. On the other hand, there might be an exception in the case of *impotentia copulandi* combined with *potentia seminalis*, where the wife submits to artificial impregnation. Here the propagation of the race would have been effected and the law would not interfere, on the ground that the parties, or either of them, could not gratify their desires, or, more strictly expressed, have intercourse successfully. In some European codes a proved sterility of both spouses affects their right to adopt a child; and this sterility is conclusively presumed after both are over fifty years of age, the marriage being childless.

Physical incapacity may be due to psychic causes, yet must not be a mere mental incompatibility. This seems somewhat paradoxic, and, in fact, may involve very difficult considerations. In one case the spouses may have made a *bona fide* attempt to consummate the marriage physically and may have failed, yet so far as the best observation can note, no physical obstacle exists. In the other case, repugnance to the sexual act may prevent fair attempt at intercourse.

The impotence must be unknown to the plaintiff at the time the marriage was contracted. Unless it is a patent case, some time must be allowed to establish a presumption of its existence. And, again, too great a time must not elapse, nor plaintiff sleep on his or her rights, nor refuse means of cure, as already stated. Ratification of the contract and waiver of grounds for avoiding it will be presumed from the *laches* of a party, as in other contracts.

An impossibility of *potentia copulandi* in a proper manner may arise from many causes—the presence of a loathsome disease, for instance. Mere age or merely having passed the menopause does not establish a good cause, as a rule. These facts must necessarily have been known or reasonably expected before marriage; besides, in neither case is there necessarily an *impotentia copulandi*. Again, there may be such a

[1] Dean *vs.* Aveling, 1 Rob. Ecc., 279.

[2] Not that gratification of desire is the exact expression of this object, since there may be successful intercourse without such gratification on both sides.

variety of hermaphroditism among its many varieties as to prevent proper copula. As to this, it seems that both sexual functions can never occur simultaneously in the same individual in the same degree, one sex predominating to the exclusion of the other, and generally to such an extent that there are only curious minor and unimportant indications of double sexuality not interfering with the plainly predominant function.[1]

In the husband the causes of *impotentia copulandi* may be, in addition to the ordinary cause of a flaccid organ impossible of erection, the shortness or amputation of the penis; the non-descension, congenital absence, or disease of the testicles; disease of the penis; large obstructions from hydrocele or hernia.

As to the natural or artificial shortness of the penis: emission may be perfect in such a case, and yet fecundation may not result. Under such circumstances as have been previously stated no remedy is given the wife, although the conditions may be so exaggerated that no copula, in the proper sense of the word, can take place. If there is not such impregnation, then in the case under consideration the question arises as to whether the degree of penetration that is possessed by the husband is such that the wife should be satisfied and not be permitted to avoid the marriage. In some of these cases it is patent that the sexual embrace is a mockery so far as the wife is concerned, although there may be erection and enjoyment on the part of the husband. Certainly in such cases, if ever, the wife would not be required to submit to artificial impregnation. This operation, it should be noted, is but rarely successful.

The non-descent of the testicles does not necessarily imply impotence; nor in case of castration is *potentia copulandi* impossible. Yet it is a question whether a husband could successfully defend a suit if such ground existed. The claim that *potentia seminalis* can exist in castration may be passed over without comment. As to what disease of the testicles exists, and whether the extent to which they are diseased constitutes impotence, only a consideration of each individual can establish. The ordinary cases of impotence[2] due to sexual exhaustion, perversion, masturbation, excess, overwrought desires, hypochondriasis, absence of desire, preoccupation, and other psychic causes may be cured. The plaintiff (wife) has a right to demand as an alternative either that the defendant (husband) attempt the cure, or, if he refuses this, the remedy given in the jurisdiction for impotence. What the effect would

[1] According to Brouardel, a genuine hermaphrodite is a myth. Hermaphroditism, so far as it relates to the internal sexual organs, is found. But the coexistence of the external attributes of both sexes is impossible. During the first six weeks of intra-uterine life the fetus is of both sexes. But the subsequent simultaneous development of the Wolffian bodies and of Müller's ducts and their accessories as yet has never been met with. In the majority of hermaphrodites the tendency is unquestionably toward the male type.

[2] See Hammond, *Impotence in the Male*. Although Hammond (p. 9) distinguishes sterility in the male from impotence, yet of the four kinds of medical impotence, three—namely, absence of sexual desire, absence of the power to ejaculate, and absence of pleasure—are not legal impotence. The fourth, absence of power of erection and of consequent intromission, being the only form that constitutes legal impotence.

be in law of an experiment upon the husband, which by the use of aphrodisiacs would result in fecundation, is very doubtful.

In the wife, impotence may be due to absence or restricted depth[1] of the vagina, such a case of absence of the uterus as makes the copulation incomplete,[2] a thick, firm hymen, atresia vaginæ, extreme displacement of the uterus, inflamed condition of the mucous lining, abnormal quantity or quality of the secretion, extreme sensitiveness of the organs (vaginismus), elongated clitoris, fibromata or other neoplasms, exaggerated nymphae, and other malformations. Some of the above obstacles may be cured. The courts expect a submission to reasonable and not degrading remedies, experiments, or operation; or the same alternative as in the case of the husband noted above.[3] An elongated clitoris may be amputated, and the hymen perforated; even a pseudovagina may be made by surgery. The operations have one aim—the possibility of copula.

In the event of impregnation, however, there is a legally conclusive presumption of sexual potency; this is so even though the hymen may be but slightly perforate, and without regard to malformations that may exist in whatever degree. In case of a known absence of the essential sexual organs except the vagina (complete ovariotomy or hysterectomy), the court might give relief on the sole ground of barrenness, in spite of the *potentia copulandi*, although no case of this sort has come under the writers' notice. On the other hand, certainly no amount of proof of barrenness will affect the marital status of the wife.[4]

All that the husband has to prove in alleging the wife's impotence is, not a general incapacity, but her incapability to receive his embrace properly. Both her general capacity and incapacity are immaterial. As with the husband, so with the wife; not only coition, but a *proper* coition is expected without need to resort to indelicate remedies. The same provision as to waiver and ratification obtains here as in the case already mentioned.

With reference both to husband and wife, the foregoing will show what care is necessary to be exercised in the use of the terms sterility, impotence, absence of desire, and barrenness. In this connection it may be said that while impotence is far more frequent among men, one woman in eight is barren, and when a marriage is sterile, the woman is to blame nine-tenths of the time.

In studying and reporting on physical incapability, the following

[1] G. *vs.* G., 33 Md., 301. [2] Dene *vs.* Aveling, 1 Rob., 274.

[3] Dohrn reports an interesting case. An individual was brought up as, and was considered, a girl. When at the age of twenty, she began to suffer from a sensation of bearing down every four weeks. Her mother sought medical aid. The physician told her that there existed no impediment to menstruation, but that if she wished to marry an incision would have to be made. The girl (?) married, and her husband shortly afterward sent her to Dohrn for examination, and he pronounced the individual a male. The couple obtained a divorce.

[4] Among the Israelites and Romans the idea of shame and disgrace went with sterility, and the latter was sufficient for the dissolution of the marriage relation. The Christian idea of marriage, as being a relation entered into for more ideal purposes, has ameliorated these opinions of sterile marriages and has taken away their severe consequences.

are the guiding questions: Is the physical peculiarity in this case of one spouse as regards the other spouse at present a total (1–2) or a partial impediment (3–4)? Is it curable or incurable (5–6)? Is it discoverable by inspection or by expert inspection, or by further experiment of cohabitation between the parties alone? Theoretically, the complaining party is sexually potent. Thus, in the old pleading, the wife as complainant alleges she is "*virgo apta* and *intacta*." But the burden of proof always rests upon the complainant.[1]

Mental Incapacity (Insanity).—An insane person, or one so deranged as not to understand the ordinary affairs of life, is incompetent to marry. Without mental capacity to give the consent necessary to a valid contract there can be no marriage. The party must be insane at the time of the ceremony or contract in order to render the marriage void; otherwise it will be held valid. If the mental incapacity were only temporary, and if the person consent to the union during subsequent lucid intervals, the marriage will be confirmed and valid.[2] It hardly seems necessary to add that the degree of insanity to render a marriage void must be such as to render the party incapable of understanding the meaning of the act: it must be more than mere weakness of intellect or natural eccentricity. The consent of a person who is markedly under the influence of liquor is good and binding, unless the degree of intoxication is sufficient to cause complete unconsciousness or mania a potu, or the drunkenness has been produced for the purpose of obtaining consent. Cases of incapacity in matrimonial actions are often, in fact, confused with and hard to distinguish from cases of fraud, mental duress, mental weakness due to age, sickness, or drugs, and particularly undue influence. All these equally may be causes for such suits. A clear line, however, should be drawn between cases of pure incapacity and those cases in which the will and intentions of a party to the marriage contract, normally competent,—although perhaps barely so,—are overcome by force of the superior personality or will of another person with or without various assisting circumstances. The connection of duress with hypnotic control should be referred to in this connection as a subject that has developed through numerous experiments of experts in this interesting field.

In a matrimonial suit founded on mental incapacity a previous adjudication of insanity, under either a writ of *de lunatico inquirendo* or otherwise, constitutes merely a rebuttable presumption.[3] Although mental incapacity, in all the types known to the nervous specialists, may be cause for the relief already indicated, whether due to disease or defect; whether temporarily manifested or artificially produced; or whether the will has been cheated or overcome by another will, still there remains the question, What form, what degree, of these general causes is required in such cases?

In analogy with cases affecting other contracts, wills, or questions of intent of a defendant at the criminal bar, the capacity required for con-

[1] 93 Ill., 373. [2] Hag. Ecc., 355; 28 Ala., 565; 23 Miss., 410.
[3] Banker *vs.* Banker, 63 N. Y., 409; 1 Hag. Con., 416.

tract by the marital relations must be the capacity to know the nature and consequences of the act done.[1] This capacity, therefore, must be *quod hoc*—*i. e.*, marriage. A marriage by one *non compos*, in order to be annulled or given the relief of the special jurisdiction, must be such as to affect the contract. That which would disqualify a person from entering into property relations might not reach his capacity to contract a marriage.[2] The law, therefore, does not deem a marriage contracted during a "lucid interval" as a subject for its interference. In general no relief can be obtained for attacks of insanity before, followed by permanent insanity after, marriage. Persons afflicted with monomania, pyromania, kleptomania, or dipsomania may not be in any legal manner unfit to make the marriage contract.

Intoxication might well legally incapacitate a person from contracting marriage at the same time that he was alive to ordinary property relations. The artificial means of producing mental incapacity are chloral, opium and its alkaloids, hasheesh (cannabis Indica), ether, and nitrous oxid. These agencies also figure in cases of unconscious adultery; the case of sexual relations, real and imaginary, resulting from the administration of nitrous oxid are quite curious. In a case where it is clearly proved that the defendant was insane at the time of the commission of adultery, no legal adultery can be made out, intent being absent.

With regard to undue influence we may well quote Wharton and Stillé, vol. i., p. 13: "Undue influence is not merely mental debility; it is the latter, plus the active, interference of another—*i. e.*, it is not subjective, but objective. Mental debility alone would not suffice. For it is conceivable that a person weak-minded to a certain degree might contract a lawful marriage, knowing the incidents of such well enough, yet a marriage of the same person in a case where another's will was imposed upon him by force of any association, suggestion, or fraudulent ingredient—not amounting alone to fraud—would be voidable."[3]

Duress.—A compulsory marriage is illegal unless afterward confirmed by the acts of the parties.[4] A man or woman forced into the relationship from fear of bodily harm is not legally married unless he or she afterward acquiesces by *voluntary* sexual intercourse or by expressed words.

In urging condonation against a wife who is plaintiff in a divorce suit, duress of the husband, that was formerly so frequently implied by law, is still considered. Thus, condonation is less readily proved of the wife than of the husband, owing to the usual relations between the sexes from a financial, social, and general historic standpoint. Of course, an insane person cannot condone.

Other Questions.—**Cruelty.**—The evidence of actual cruelty is the same as that of ordinary assault cases, as to marks of violence, etc.,

[1] 52 Me., 505; 44 Nft., 531; 14 Rob., 488; 24 Wend., 85; 1 Houst., 308 (Delaware); 55 Me., 256; 4 Con., 217; 21 Wend., 142.
[2] True *vs.* Ranney, 1 Fost., 4
[3] Undue influence: Perkins *vs.* Scott, 23 Iowa, 237; Portsmouth *vs.* Portsmouth, 1 Hag. Ecc., 355
[4] 6 Baxt., 297.

treated elsewhere. But in matrimonial actions—cruelty being a cause for divorce in some jurisdictions, and for a separation in others—there are, in addition, many and differing interpretations of the term cruelty. Thus, under this head, are fear and mental cruelty. Further, cruelty is a relative term, "*versus hanc*," so that the circumstances of each case are separately considered with relation to the relative power of inflicting, and susceptibility to, either physical or mental torture.[1] Again, in jurisdictions where sodomy or sexual perversion generally is not a cause of divorce, it may constitute "cruelty" or cause for separation.

Both refusal of the sexual embrace and the infliction of excessive intercourse have been held to be cruelty. A venereal disease communicated to the plaintiff by the defendant has been so considered. Profanity, various kinds, degrees, and amounts of abuse, either with or without threats, have been held cruelty: whether produced by insanity or disease, it is cruelty; the innocent spouse need not suffer it. The question of *intent* not being considered, it does not avail the defendant to show his cruelty to be due to temper and not to malice. The health, temperature, and mental condition of the plaintiff being of such importance, the physician and nerve-specialist have an ample field in these cases, and are actually employed largely on them. Conduct that causes great mental suffering, making life unbearable, very frequently has been held sufficient.[2]

In cases of mental cruelty the real theory is that the mental condition has had, or would have had, an indirect effect on the physical.

Evidences as to unchastity, of unnatural practices, of venereal disease, of pregnancy, of rape, of copula, are the same, as far as the questions bear on marital questions, as in the other connections elsewhere discussed.

In a unique case coming under the direct observation of the authors, and in which they were professionally employed, evidence of menstruation was given, in a case of divorce for adultery, as constituting or tending toward a defense. As the defendant was found almost *in flagrante delicto*, the plaintiff very properly considered that this evidence was not highly presumptive of innocence and refused to cross-examine or rebut as to such a class of evidence. If it is claimed that a forced marriage has been even physically consummated by force, evidence as to rape becomes relevant.

Copula.—Disaffirmance of marriage with voluntary copula is generally necessary to get relief for fraud. Copula under an informal oral contract to marry is necessary to complete such marriage. Note in this connection the expression, "cohabited for any considerable period of time" in the New York laws.

Pregnancy.—Concealed pregnancy at time of entering into the marriage contract is a ground for annulment; otherwise, in general, even concealed antenuptial pregnancy is no ground for annulment. This rule is founded on favor shown marriage by public policy.

[1] No law of averages, of the normal effect of the conduct complained of, on a normal person is considered.

[2] 44 Ala., 670; 18 Kan., 419; 18 Texas, 226; 31 Wis., 235, etc.

Venereal Disease.—This may be the basis of a charge of cruelty, and may be evidence of adultery if fastened upon the guilty party too long a time after the beginning of the marriage to be the result of intercourse before marriage.

Access, Birth of Child (As stated elsewhere more fully under Birth and Legitimacy).—A child who could have been born within a legal time after access is presumed to be the child of the husband; but if without such time, is illegitimate and is proof positive of adultery and a cause for divorce.

Unchastity.—Concealed antenuptial unchastity, unless pregnancy result and exist at marriage, is almost without exception insufficient cause for annulment. The rule here on public grounds is, *caveat emptor*. Having been, unknown to husband, a prostitute before marriage is a ground, however, in some jurisdictions, as in Virginia and West Virginia.

Access.—In general this equals cohabitation in contemplation of law. To all intents and purposes they are synonyms. This is particularly important in questions of condonation. The presumption is but slightly stronger in a case where spouses stay all night in a room where there is but one bed, than in such a well-contrasted case where they are merely alone for a few minutes in the same room by day, or have privacy for a short time in a separate domicile (private house or apartment), using a suite of rooms and servants being in such domicile.

Miscegenation.—Before leaving the topic of marriage and divorce, the subject of miscegenation should not be omitted. In several of the states, as already stated, marriages between white persons and negroes or Indians are strictly forbidden under penalty, the marriage being void or voidable according to the jurisdiction. The ground for this legislation is the public health. It is based upon physiologic facts and theories, and the general belief that the natural law of selection, "each after its own kind," applies to races of men as it does to animals. Hybrid or cross breeds are regarded as inferior to the pure blood of either race. A famous southern Methodist bishop (colored), however, shows, by appeal to facts and figures, that the African race on this continent is becoming rapidly absorbed by and into the Caucasian or white race by irregular unions without any appreciable deterioration. From this it appears that the apprehension of demoralization, etc., is not well founded so far as miscegenation between negroes and whites is concerned. Still, where such prohibition is in vogue, there should be a severe penalty for those persons who deliberately and illegally form a union to bring into the world children whom they know can never become legitimate. The innocent little sufferers of such marriages are chiefly to be considered, and the law aims at prevention by inflicting heavy penalties; while by merely declaring the marriage void, the greatest punishment falls directly upon the children, and not upon the parties themselves.

Mental Incapacity.—This is sometimes a ground for matrimonial actions, annulment, divorce, or separation, according to the jurisdiction, as has been noted, and is also a defense to civil and criminal charges

of adultery. As a cause of such actions the insanity must, in general, exist at the time of making the marriage contract; incurable chronic mania or dementia for ten years—*i. e.*, after marriage—is, however, a cause for divorce in the state of Washington; and insanity after marriage, for limited divorce or separation in Arkansas. The insanity must be concealed from the complainant, and the latter must not delay his application for a remedy or act so as to ratify the marriage. Antenuptial temporary insanity is, as a rule, insufficient. Mental incapacity as a defense to charges of adultery is one of the evidences of lack of intent without which a case of this offense cannot be made out.

Logically, incapacity at the time of marriage should be a ground for a suit to annul and not for divorce. Insanity as a defense in adultery became an important question in the celebrated Mordaunt case, in which King Edward VII. was named as corespondent.[1] In the case of an insane defendant the consequences are sufficiently terrible, whether testimony of such defendant's guilt is admitted or refused.

[1] See Ordman's *Judicial Aspects of Insanity*, p. 339.

MALPRACTICE.

MALPRACTICE may be defined to be the failure, to the damage of the patient, on the part of a physician or surgeon to exercise that degree of care, skill, diligence, and judgment that the law requires in the treatment of such patient.

Malpractice may be considered under two heads: civil and criminal.

1. Civil Malpractice.—The gist of an action for civil malpractice is negligence. The rules of law governing this important subject are well stated by Judge Cooley in his work on *Torts*. We cannot do better than quote his language:

"As the promise is not different in the case of the physician and surgeon from what it is in the case of an attorney, solicitor, and proctor, one general rule may be given which will apply to all.

"The English authorities are, perhaps, somewhat more indulgent to the faults and mistakes of professional men than are those of this country. Thus, Lord Campbell, with the full concurrence of his associates in the House of Lords, declared that in order to maintain an action against one's legal adviser, it was necessary, 'most undoubtedly, that the professional adviser should be guilty of some misconduct, some fraudulent proceeding, or should be chargeable with gross negligence or with gross ignorance. It is only upon one or the other of these grounds that the client can maintain an action against the professional adviser.'

"On the other hand, the rule is laid down in Pennsylvania that the professional man must bring to the practice of his profession a degree of skill and diligence such as those 'thoroughly educated in his profession ordinarily employ.' This is a severe rule, and fixes a standard of professional skill and attainments which, in the newer portions of the country, would be quite out of the question. In New Hampshire the undertaking of the practitioner has been stated in the following language: 'By our law a person who offers his services to the community generally, or to any individual, for employment in any professional capacity as a person of skill, contracts with his employer: 1. That he possesses that reasonable degree of learning, skill, and experience which is ordinarily possessed by the professors of the same art or science, and which is ordinarily regarded by the community and by those conversant with that employment as necessary and sufficient to qualify him to engage in such business. 2. That he will use reasonable and ordinary care and diligence in the exertion of his skill and the application of his knowledge to accomplish the purpose for which he is employed. He does not undertake for extraordinary care or extraordinary diligence, any

more than he does for uncommon skill. 3. In stipulating to exert his skill and apply his diligence and care, the medical and other professional men contract to use their best judgment.' This is believed to be an accurate statement of the implied promise. The practitioner must possess at least the average degree of learning and skill in his profession in that part of the country in which his services are offered to the public; and if he exercises that learning and skill with reasonable care and fidelity, he discharges his legal duty."

A different rule has been at times laid down. Thus, in McCandless *vs.* McWha, 22 Penn. St., 261, the rule was stated to be that such skill and diligence must be exercised "as thoroughly educated surgeons ordinarily employ"; and in Holtzman *vs.* Hoy, 118 Ill., 534, Mr. Justice Mulkey states that the duty of the defendant "was to bring to the case in hand that degree of knowledge, skill, and care which a good physician and surgeon would bring to a similar case under like circumstances." The rule first above stated is, however, believed to be the correct rule.

Negligence is never presumed, but must be proved. In an action for malpractice the burden of the proof is, therefore, upon the plaintiff to show that the defendant failed to exercise due skill, care, and diligence, and that the damage to plaintiff was the result thereof. The professional skill of the defendant is the issue in such a case; but the only way of showing a lack of it is by proving that he did not exercise it in the treatment of the plaintiff. The possession or want of proper skill cannot be proved by general reputation.

The mere fact that the defendant has failed to cure the patient raises no presumption of the want of proper care, skill, and diligence; and the fact that a fractured limb is shorter after the recovery of the patient than it was before the injury is not even *prima facie* evidence of a want of skill on the part of the attending surgeon.

One who is not a regular physician, but who holds himself out as such, is bound to exercise the skill and diligence usually possessed by regular physicians in the treatment of such diseases.

A physician possessing a reasonable degree of learning and skill, and exercising, according to his best judgment, reasonable and ordinary care and diligence, is not liable for a mere error of judgment in advising a particular remedy about which there is a difference of opinion. To hold a physician liable for every error of judgment made in the ordinary course of practice would be to debar him from the exercise of his profession and to deprive the public of the benefit of valuable service. The law does not require infallibility. A medical man cannot, as a rule, be held guilty of negligence for not employing any particular remedy, since, as a rule, there is never any one specific remedy in the use of which all authorities are agreed. If it could be shown, however, that all authorities agreed that a particular drug should be used in a particular case, as, for example, a certain antidote in a case of poisoning, the failure to employ such specific would probably constitute actionable negligence.

There is no implied warrant from the contract of employment that

a cure will be effected. It is competent, however, for a physician to contract, if he sees fit to do so, that if there is no cure there shall be no pay.

A physician may decline a case, but having once undertaken it, he must continue his services, even if gratuitous, until a reasonable time has been given the patient to procure another physician; where his services are not gratuitous he has no right to desert a patient without reasonable cause before the end of the illness he has undertaken to treat.

As respects *voluntary services*, Judge Cooley lays down the rule thus: "Where friends and acquaintances are accustomed to give, and do give, to each other voluntary services without expectation of reward, either because other assistance cannot be procured, or because the means of parties needing help will not enable them to engage such as may be within reach, the law will not imply an undertaking for skill, even when the services are such as professional men alone are usually expected to render. And where there is no undertaking for skill, the want of it can create no liability. So the 'street opinion' of an attorney, given in answer to a casual inquiry by one to whom he holds no professional relation, cannot, however erroneous, render him liable. But when one holds himself out to the public as having professional skill, and offers his services to those who accept them on that supposition, he is responsible for want of the skill he pretends to, even when his services are rendered gratuitously." Under the circumstances first above stated the one who undertakes the treatment of a patient, either voluntarily or upon request, is liable only for gross negligence; but if by forcing himself into a case he excludes a competent physician, he is liable for slight negligence or for lack of the skill and diligence of the specialist. In general it may be said that the liability of one rendering medical services is measured by the amount of skill he undertakes to exercise; and, as we have seen, the matter of compensation is immaterial.

A physician must always use his best judgment; and while he is not responsible for mere *errors of judgment* or mere mistakes in matters of reasonable doubt and uncertainty, if the error of judgment is so gross as to be inconsistent with the use of the degree of skill required by the law, he will be liable to an action. Where errors of judgment result from the want of ordinary care and skill, responsibility attaches, however carefully the judgment is exercised. In exercising his best judgment the physician is required only to anticipate the nature and probable consequences of his treatment. It has been held that he cannot be held responsible for the disastrous effect resulting from administering chloroform as an anesthetic to a patient of a peculiar temperament where such peculiarity was unknown to him. Its administration, however, without a previous careful physical examination of the patient, or its administration by a dentist without the assistance of a competent physician, and to a patient in an upright position, seems clearly culpable. Common prudence would also require that neither it nor any other anesthetic should be administered without the aid of a competent physician.

A physician cannot lawfully try experiments upon his patients to their injury.

One who professes to adhere to a particular *school of practice* must come up to its average standard, and must be judged by its tests and in the light of the present day.

As to the mode of treatment in a given case, when it conforms to the settled practice of the particular school to which the physician belongs, he is relieved from all responsibility; in such case, evidence of the practice of physicians of other schools is inadmissible. Where, however, the case will admit of but one mode of treatment, the use of a different mode would be evidence to show a want of skill..

According to the well-considered case of Nelson *vs.* Harrington, 72 Wisc., 591, to constitute a school of medicine within the rule relieving from liability in cases of alleged malpractice, it must have rules and principles of practice for the guidance of all its members, as respects principles, diagnosis, and remedies, which each member is supposed to observe in any given case. So-called clairvoyant physicians, who have no recognized method of treatment, were in this case held not to constitute a school of medicine within the above rule; and hence such an alleged physician cannot escape liability for malpractice by showing that he treated the plaintiff with the ordinary skill and knowledge of the so-called clairvoyant system. To escape liability he must show that he treated him with the ordinary skill and knowledge of physicians in good standing practising in that vicinity.

The proper and only mode of showing want of skill on the part of the defendant is, as above stated, by proving that he did not exercise it in the particular case. It can neither be established nor disproved by showing the defendant's general professional reputation; it is improper, therefore, to ask a witness what the reputation of the physician is in the community and among the profession as being an ordinarily learned and skilful physician. The treatment of each individual case is the criterion for ascertaining the physician's liability. It has been held, also, that the general opinion of the practitioner with whom the physician studied his profession, or of the professors of the school at which he was graduated, is inadmissible.

A sign or other proof that one actually practises medicine or surgery is *prima facie* evidence of his professional character. The possession of a medical diploma, issued by a college having authority to grant degrees in medicine, is *prima facie* evidence of ordinary skill.

The mere fact that the defendant refused consultation with other men of his science is no assumption upon his part that he is possessed of more than ordinary skill; and his declination in this respect does not vary the application of any of the rules above stated.

As to the proof of alleged malpractice in the treatment of an injured limb, it is generally proper to allow the plaintiff to exhibit the limb to the jury; it has been held, however, that the limb upon which the alleged malpractice was practised cannot be exhibited to the jury if several years have elapsed since the injury.

As to whether the plaintiff in an action for malpractice or other injury caused by negligence can be compelled to submit his person to an examination by physicians before the trial is a question involved in great conflict. The better opinion seems to be that, independent of statute, courts of common law have no such power.[1]

It is the duty of the patient to coöperate with the physician in his endeavors to effect a cure. If the injury complained of is due to the contributory negligence of the plaintiff, no recovery can be had; if the want of coöperation of the plaintiff merely aggravates the effect of malpractice on the part of the physician, it will not debar a recovery, but will mitigate the damages. Negligence of the nurse concurring with that of the physician has been held to be imputable to the patient. This, however, will depend upon the question whether such a nurse was the agent of the patient or physician. If the acts of negligence can be so separated as to show that the injury was due solely to the fault of the physician, and the fault of the nurse was only remotely connected therewith, an action will lie against the physician.

A physician is responsible for the results of his negligence or unskilfulness notwithstanding the case is given over by him to another; of course, however, he will not be responsible for the subsequent negligence of the physician to whom the case is transferred. If a family doctor, or the surgeon of a company or society, on leaving home recommends, in case of need, some other physician, who is not, however, in any sense in his employment, it does not make him in any way liable for the injuries arising from the latter's want of skill.

In an action brought by a father against two medical men to recover damages for advising him to suck a tracheotomy tube of his child, who had just been operated on for diphtheria, without first duly warning him of the risk he ran, whereby he himself became infected with the disease, it was held that there was no cause of action.

The capacity of the patient injured to judge of the probable results is an important element in cases of malpractice; hence, if the patient is insane, he cannot be chargeable with contributory negligence; but where a patient relies upon his own judgment and not upon that of the surgeon, as to the propriety of the operation, it is held that the surgeon is not liable for the injurious consequences resulting therefrom, premising, of course, that due care and skill are exercised in performing such operation.

If a person is attacked by a fatal disease and there is no escape from

[1] See McQuigan *vs.* Delaware, etc., R. R. Co., 129 N. Y., 50, S. C. 29 N. E. Rep., 235; Union Pacific Railroad Company *vs.* Botsford, 141 U. S., 250; Parker *vs.* Enslow, 102 Illinois, 272; Loyd *vs.* Hannibal and St. Joe Railroad, 53 Missouri, 509.

Contra, Schroeder *vs.* Chicago, etc., Railway, 47 Iowa, 375; Miami, etc., Company *vs.* Bailey, 37 Ohio St., 104; Atchison, etc., R. R. Company *vs.* Thul, 29 Kansas, 466; White *vs.* Milwaukee Railway, 61 Wisconsin, 536; Hatfield *vs.* St. Paul, etc., Railroad Company, 33 Minnesota, 130; Stuart *vs.* Havens, 17 Nebraska, 211; Owens *vs.* Kansas City Railroad Company, 95 Missouri, 169; Sibley *vs.* Smith, 46 Arkansas 275; Missouri, etc., Railroad Company *vs.* Johnson, 72 Texas, 95; Richmond, etc., Railroad *vs.* Childress, 82 Georgia, 719; Alabama, etc., Railway *vs.* Hill, 90 Alabama, 71.

it save by a dangerous surgical operation, then if he gives his free and intelligent consent to the operation and it is skilfully performed, the surgeon cannot be blamed, even though the patient perishes under the knife. The burden of proof that the patient did not consent to the operation is upon the plaintiff. If a woman in labor is in such a condition that her life can be saved only by the sacrifice of that of the child, then it is not only the right, but the duty, of the attendant to save the mother at the expense of the child. Of course, in such a case counsel should be had when possible before resorting to such extreme measures.

The burden of showing that the use of instruments to produce abortion was necessary to save the life of the woman is on the defendant.

The rules of law above stated respecting the liability of physicians for negligence and malpractice are equally applicable to similar charges made against midwives, nurses, medical students, chemists, pharmacists, and against any other person who holds himself out as possessing knowledge or skill in any particular department of learning or practice.

Dentists likewise are subject to the same rules as to negligence as physicians and surgeons. A patient must exercise ordinary care and prudence; so that if one tells a dentist to pull out a tooth, but does not say which one should be pulled, and the wrong one is taken out, the sufferer has no legal ground of complaint unless, indeed, it is quite apparent which is the offending member. The patient may have been a little careless and negligent; still, if the dentist has been so very neglectful of his duty that no ordinary care on the part of the patient would have prevented the mistake or injury complained of, the injured party may recover damages. The fact that one has taken chloroform will not affect his rights or remedies against a dentist for any mistake or negligence.

The fact that a dentist extracts teeth gratuitously does not relieve him from liability for failure to perform his work properly. As in the case of physicians, so in the case of the dentist, it is a good answer to an action to recover payment for his work and labor that the defendant has been injured, instead of benefited, by the plaintiff's treatment, either because of his negligence or want of skill.

Sex is no excuse for negligence: there is no rule of law that less care is required of a woman than of a man.

If a physician should be so indiscreet as to make a special contract to cure a person of a certain disease or deformity, or to bring about any other desirable result, he will, of course, be liable for damages for a breach of contract if he fails to perform his agreement. Common prudence would, therefore, dictate great caution in this respect, and a guarded prognosis in every case, lest the patient may pervert what is intended as a mere prognosis into a positive engagement to cure.

Although where the statute of a state requires a state license in order to authorize a physician to practise medicine or surgery, an unlicensed physician cannot recover at law compensation for services rendered by him, the fact that he is unlicensed does not affect his liability to an action for malpractice.

Although it may not afford much consolation to the defendant in a malpractice suit, the rule of law is that an action for malpractice does not at common law survive the death of the defendant, and hence does not, in the absence of a statute to the contrary, constitute a claim against his estate.

Although it is not within the power of a state legislature to discriminate in favor of any particular school of medicine, yet such laws may be enacted as will protect the people from ignorant pretenders, and require learning and skill in the school of medicine which the physician professes to practise. No school of medicine is exempt from liability to an action for malpractice. Where one not specially qualified volunteers to heal a sick person, he can be required to bestow only such skill and diligence as are usually exercised under such circumstances by persons of like qualifications. If a patient voluntarily employs in one art a man who openly exercises another, his folly has no claim to indulgence. The old Mahomedan case cited by Puffendorf with approbation is very much to the point: A man who had a disorder in his eyes called on a farrier for a remedy, who gave him one commonly used upon his quadrupedal patients. The man lost his sight and brought an action against the farrier for damages, but the judge held that no action would lie, for if the complainant had not himself been an ass, he would never have employed a horse-doctor.

But, on the contrary, it has been held that an expert in the diseases of man is necessarily an expert in the diseases of animals, so as to make his opinion competent evidence upon the question as to whether a disease with which a mule was afflicted was of recent origin or of long standing.

However much a regular physician may differ from the method of treatment adopted by the profession of any other school, such as homeopathy or hydropathy, these systems have acquired a certain recognition by the public, and in some instances legal recognition by statute; and it is not to be expected that an adverse verdict will be given merely because the medical attendant *pro tempore* practised according to these systems. It is a well-known fact that the same drugs are to a very considerable extent used by the best practitioners in the two leading schools of practice; and so far as concerns the practice of surgery and obstetrics, the general principles of practice must necessarily be the same in every rational system. On the other hand, mesmerism, Coffinism, Morrisonism, have never acquired such a status, and are not likely to do so. Certain other systems of charlatanism which at present are occupying an undue share of the attention of the more credulous portion of the community, such as so-called "clairvoyance," "Christian science," "faith healing," and "metaphysics," should be included in the same category. It would, as it seems to us, be very difficult to frame a defense to a charge of malpractice preferred against one professing to heal certain ailments by either of the last four so-called systems. Suppose the case, for instance, of the treatment of strangulated hernia, or a compound fracture, dislocation, placenta prævia, or hemorrhage

from a divided artery, by means of faith or so-called metaphysics, or any other like inefficient means. It would not be possible, in our judgment, to frame a defense which would relieve a party practising such or any other mere expectant treatment from liability for malpractice.

As to the question of *doses*, however, even in so-called orthodox medicine, very considerable latitude within certain extremes must necessarily be allowed. Common sense and experience are the only safe criteria in such case. As is well observed, however, by Drs. Woodman and Tidy, "No experience and no theory can be held to justify giving an infant only a month old ½-grain doses of opium at frequent intervals as a dental sedative, or ¼-grain doses of strychnin as a tonic."

Several rules may be found in treatises upon therapeutics by which to determine the dose proper for patients under adult age.

Young's rule is to add 12 to the age of the patient and divide the age by the sum. Thus, a child one year old would require $\frac{1}{13}$, and one three years old $\frac{3}{15}$ $(\frac{1}{5})$ of the amount necessary for an adult.

By *Cowling's rule* the proportionate dose for any age under adult life is represented by the number of the following birthday divided by 24. Thus, for a child one year old the dose would be $\frac{2}{24}$ $(\frac{1}{12})$ of that for an adult.

Clarke's rule is based upon relative weights. Assuming the average weight of an adult to be 150 pounds, for whom an appropriate dose is unity, the dose of most medicines must be increased or diminished in the proportion of the weight of the patient to that number of pounds. This proportion is represented by a fraction whose numerator is the patient's weight and whose denominator is 150.

Actions for malpractice are most frequently brought where an unfavorable result has followed the reduction of a fracture or dislocation or some other surgical operation. It would be interesting to review the reported cases wherein these particular acts of malpractice are more fully discussed, and also to state the leading principles of the practice of medicine and surgery bearing upon such cases; but the limits assigned to this article forbid entering into such details. For further details reference is made to the cases cited in the various digests of reported cases, and to the *Compilation of Reported Cases upon Civil Malpractice* by Dr. McClelland, in which latter book will be found a large number of cases of malpractice in the various departments of medicine, surgery, obstetrics, etc.

2. Criminal Malpractice.—Bishop, in his work,[1] lays down the rule as to homicide from carelessness thus:

"Every act of gross carelessness, even in the performance of what is lawful, and *a fortiori* of what is not lawful, and every negligent omission of legal duty, whereby death ensues, is indictable either as murder or manslaughter.

"If a man take upon himself an office or duty requiring skill or care,—if, by his ignorance, carelessness, or negligence, he cause the

[1] *Criminal Law*, vol. i., sec. 314, seventh edition.

death of another, he will be guilty of manslaughter. . . . If a person, whether a medical man or not, profess to deal with the life or health of another, he is bound to use competent skill and sufficient attention; and if he cause the death of another through a gross want of either, he will be guilty of manslaughter."

Bishop says further:[1]

"The doctrine as to physician and patient is not quite the same in England and the United States. And possibly it is not entirely harmonious among our states. According to English adjudication, whenever one undertakes to cure another of disease or to perform on him a surgical operation, he renders himself thereby liable to the criminal law if he does not carry to this duty some degree of skill, though what degree may not be clear; consequently, if the patient dies through his ill treatment, he is indictable for manslaughter."

"Still, in an English case," says Bishop in the same section, "Willes, J., once put the doctrine in a more reasonable way, thus: 'If a man *knew that he was using medicines beyond his knowledge*, and was meddling with things beyond his reach, that was culpable rashness. Negligence might consist in using medicines in the use of which care was required and of the properties of which the person using them was ignorant. A person who so took a leap in the dark in the administration of medicine was guilty of gross negligence.'" Bishop then very characteristically and dogmatically observes: "Now, in the facts of human life, the less a man understands of anything occult, like the unseen workings of medicine, the more confident he is that his knowledge of the thing is perfect. Therefore, some of our American courts have laid down the doctrine, not altogether inharmoniously with this utterance of the learned English judge, in substance that, since it is lawful and commendable for one to cure another, if he undertakes this office in good faith and adopts the treatment he deems best, he is not liable to be adjudged a felon, though the treatment should be erroneous, and in the eyes of those who assume to know all about this subject, which in truth is understood by no mortal, grossly wrong; and though he is a person called by those who deem themselves wise, grossly ignorant of medicine and surgery."[2]

We have quoted thus largely from Bishop's excellent book because we believe that in thus dogmatizing concerning the lack of knowledge of their profession by those following the practice of medicine and surgery, he has himself erred through gross ignorance. "If a man *knew that he was using medicines beyond his knowledge*, and was meddling with things beyond his reach, that was (indeed) culpable rashness." But it does not appear that Justice Willes, in the case from which the above quotation was made, which was from a charge to the jury at the Durham Assizes, 1864, intended to say that this was the only kind of culpable rashness. It seems, on the other hand, that this was merely

[1] *Criminal Law*, vol. i., sec. 664.

[2] *Ibid.*, vol. ii., sec. 664, citing Commonwealth *vs.* Thompson, 6 Mass., 134; and Rice *vs.* State, 8 Mo., 561. See also State *vs.* Schulz, 55 Ia., 628.

an illustration; for he immediately adds: "Negligence might consist in using medicines in the use of which care was required," etc. (See *supra.*) That it was merely an illustration is further apparent from the fact that immediately thereafter he adds another illustration: "If a man were wounded, and another applied to his wound sulphuric acid, or something which was of a dangerous nature and ought not to be applied, and which led to fatal results, then the person who applied this remedy would be answerable, and not the person who inflicted the wound, because a new cause had intervened." In the beginning of his charge the learned judge very properly said: "Every person who dealt with the health of others dealt with their lives, and every person who so dealt was bound to use reasonable care and not to be grossly negligent. . . Another sort of gross negligence consisted in rashness, where a person was not sufficiently skilled in dealing with dangerous medicines which should be carefully used, of the properties of which he is ignorant, or how to administer a proper dose. A person who, with ignorant rashness and without skill in his profession, used such a dangerous medicine, acted with gross negligence." The drug given in this case, and which caused death, was a tablespoonful of a tincture of colchicum seeds, containing 80 grains of the seeds, 18 grains, as is said in this case, being a fatal dose.

In *Nanny Simpson's case*[1] the prisoner was indicted for manslaughter in having caused the death of a man by administering *white vitriol* as a medicine. Bailey, J.: "I am clear that if a person not having a medical education, and in a place where persons of a medical education might be obtained, takes on himself to administer medicine which may have a dangerous effect, and such medicine destroys the life of the person to whom it is administered, it is manslaughter. The party may not mean to cause death; on the contrary, he may mean to produce beneficial effects; but he has no right to hazard medicine of a dangerous tendency where medical assistance can be obtained. If he does, he does it at his peril." See also *Tassymond's case*,[2] where the prisoner was convicted of manslaughter in causing the death of an infant by negligently selling laudanum for paregoric.

It may be conceded that the cases of Commonwealth *vs.* Thompson, 6 Mass., 134 (decided in 1809), and Rice *vs.* State, 8 Mo., 561 (decided in 1844), in the former of which the law of the case is contained in Chief Justice Parson's charge to the jury which tried the prisoner, and the latter of which is apparently decided mainly upon the authority of the former, seem to lay down the rule that in order to warrant a conviction for murder or manslaughter, the defendant must have some knowledge of the fatal tendency of the prescription. An attentive perusal of these cases cannot fail, however, as it seems to us, to convince the reader that there was a palpable failure of justice in both cases.

In the case of Commonwealth *vs.* Thompson the defendant gave to a patient suffering with a cold powdered lobelia, and persisted in giving it to him for a period of eight days, until he was so completely exhausted that no relief could be afforded, and he died of exhaustion.

[1] 1 Lewin, 172, 262.

[2] *Ibid.*, 169.

In Rice *vs.* The State the defendant in the court below was employed by the husband of a woman near the end of the eighth month of pregnancy, to cure her of "sciatica"; and after having been informed of her condition and that other physicians had cautioned against the use of vapor baths and emetics in her then condition, he commenced a course of treatment by steaming and giving lobelia, and persisted in this treatment until she had a premature delivery, a few days after which she died. The evidence showed that she had been married five years, and during that time had had three children, always doing well after a confinement, and was in better health when the defendant commenced his practice on her than she had been for many years.

Commonwealth *vs.* Thompson and Rice *vs.* The State were approved and followed in the State *vs.* Schulz, 55 Iowa, 628 (decided in 1881).

After a consideration of the reported cases it seems to the writer that the cases of Commonwealth *vs.* Thompson and Rice *vs.* The State are clearly wrong. In our opinion the rule laid down in the case of Commonwealth *vs.* Pierce, decided by the Supreme Court of Massachusetts, is much more rational, and virtually overrules the case Commonwealth *vs.* Thompson. In Commonwealth *vs.* Pierce, 138 Mass., 165 (decided in 1884), it was held, Holmes, J., delivering the unanimous opinion of the Court, that to constitute manslaughter where there is no evil intent it is not necessary that the killing should be the result of an unlawful act; it is sufficient if it is the result of reckless or foolhardy presumption, judged by the standard of what would be reckless in a man of ordinary prudence under the same circumstances.

The defendant in this case, who publicly practised as a physician, being called upon to attend a sick woman, caused her with her consent to be kept for three days in flannels saturated with kerosene, by reason of which she died. There was evidence that he had made similar applications with favorable results in other cases, but that in one the effect had been to blister and burn the flesh, as in the present case. It was held that the jury having found that the application was made as the result of foolhardy presumption or gross negligence, a conviction of manslaughter was proper.

The Court in the case of Commonwealth *vs.* Pierce has carefully limited the application of the rule there laid down to cases where there was no sudden emergency and where no exceptional circumstances were shown; and thus limited, the rule of the case seems eminently reasonable and grounded on the soundest views of public policy.

THE MEDICOLEGAL RELATIONS OF THE RÖNTGEN OR X-RAYS.

THE forensic importance of the Röntgen rays relates to the admissibility of skiagraphs in evidence, to their use in surgery, and to certain untoward results which may follow their application.

As yet, comparatively few cases have reached the higher courts, as the epoch-making discovery of Röntgen was not announced until December, 1895. Our knowledge of the exact nature of the Röntgen rays has increased but little since the discoverer made his original announcement to the Wurzburg Physico-Medical Society. Not fully understanding the nature of the phenomenon, he called them *x*-rays. Their properties, as far as they have been studied, show that they proceed in a straight line and cannot be refracted or difracted, and can be reflected only to a small extent. Matter is opaque to these rays in proportion to its atomic weight, the lighter allowing more of the rays to pass and the heavier interrupting the rays and casting shadows. They act upon a photographic plate like light, and cause fluorescence of certain crystals, the best for this purpose being platinocyanid of barium and platinocyanid of potassium.

The rays are obtained by exciting a tube, which may be done by connecting it with a coil or with a static machine. The latter has been claimed by some to give better results and to be less liable to burn the skin. This is not correct, as several cases of severe burning have been reported in which a static machine had been employed. Burning relates more to the tube than to its method of excitation.

There are many tubes upon the market, varying in their construction. In a good tube the rays should come from a fine point on the anode, and there should be an arrangement for reducing the vacuum if it gets too high. To work successfully with the *x*-rays requires that one should have several tubes with high and low vacuum, the latter being employed in work upon the extremities and for therapeutic purposes; the former for examining the deeper-lying structures.

A method of protecting the operator from the stray *x*-rays which proceed in every direction from the point near the anode is to surround the tube with a covering to cut off these extraneous rays. Such covering should have opposite the anode, where there is a maximum of brilliancy, an opening. The most effective way is to bury the tube in red lead in a wooden box, and then cut an opening of sufficient size for the purpose required. In this way better definition is obtained when taking a radiograph, because the plate is not clouded by the extraneous rays.

In treatment this method of covering the tube will be found to be more efficient than covering the patient, as it is difficult to do the latter and at the same time protect the operator.

The admissibility of radiographs in evidence has comparatively few decisions. As a general rule, the trend of courts is to admit them, and in some cases to place them upon the same plane as photographs. Indeed, some of the higher courts, in discussing the admissibility of radiographs, speak of them as photographs. This is manifestly wrong, as they are not in any sense photographs, but are pictures of shadows. If the term "shadowgraph" had been adopted, it would have given a much better idea of the manner in which these pictures are produced, and would perhaps have given the public a better idea of their value. The danger of submitting them to a jury is that they may construe them as an ordinary photograph, and, as such, draw erroneous conclusions.

The possibility of distortion in the taking of these pictures will be recalled by the parlor-plays in which shadows are thrown upon a sheet, the sole source of light being behind the object causing the shadow. Peculiar contortions and grotesque effects are familiar to all, and the alteration that is produced in the shadows by a slight change in the position of the object or in the source of the light is well understood. The same mechanism is called into play when a radiograph is taken. If the plate is moved a slight distance from the object, there is immediate alteration of the shadows and their relations. The same is true if the tube is moved. Radiographs have shown what appeared to be fractures where none existed; in other cases demonstrable fractures have shown no signs of a break when radiographed. The callus which is thrown out about a fractured bone is very translucent and may show a space which resembles a fracture; at the same time the bone may have sufficient strength to bear the weight of the body.

A remarkable case is reported in the *Cleveland Medical Journal*, December, 1902. A gastrotomy was performed for removal of false teeth which were supposed to have been swallowed. Every diagnostic measure seemed to have been resorted to in the case, and finally an *x*-ray examination was made, which showed at the cardiac end of the esophagus a deep shadow. This was supposed to locate the teeth. The patient died as a result of the operation, and upon autopsy it was found that the shadow in the skiagraph was caused by calcareous plaques in the aorta.

A skiagraph needs to be properly interpreted, with due regard to its limitations; hence, allowing it to go to the jury without explanation may lead to an erroneous interpretation. Even surgeons who are familiar with the normal relations of joints may fail in interpreting a radiograph. Hoffmeister, in April, 1898, showed to the German Surgical Society radiographs of a normal adult pelvis which gave numerous false appearances by altering the position of the tube and plate. Those who are best skilled in estimating the value of radiographs are a unit in the opinion that they should be taken only by skilled operators, and their interpretation should be in the hands of someone who has had practical

experience in radiographic work. This is, of course, no argument against their admissibility in evidence, but simply that it should be subject to certain limitations.

Comparatively few courts have discussed the admissibility of radiographs in evidence. The First District Court of Colorado,[1] in scarcely more than a year after the announcement of Röntgen's, used the following language in respect to a radiograph that was offered in evidence:

"These exhibits are only pictures or maps to be used in explanation of a present condition, and, therefore, are secondary evidence and not primary. They may be shown to the jury as illustrating or making clear the testimony of experts."

The Court, while not one of last resort, correctly announced the rule that should govern the introduction of radiographs. In explanation of its position in the matter the Court said:

"During the last decade no science has made such mighty strides forward as surgery. It is manifestly a scientific profession, alike interesting to the learned and the unlearned. It makes use of all science and learning. It has been of inestimable value to mankind. It must not be said of the law that it is wedded to precedent; that it will not lend a helping hand. Rather let the courts throw open the door to all well-considered scientific discoveries. Modern science has made it possible to look beneath the tissues of the human body and has aided surgery in telling of the hidden mysteries. We believe it to be our duty in this case to be the first, if you please so to consider it, to admit in evidence a process known and acknowledged as a determinate in science. The exhibits are admitted as evidence."

In Bruce *et al vs.* Beall, Supreme Court of Tennessee,[2] a physician was permitted to submit to the jury an *x*-ray photograph which showed the overlapping bones of the plaintiff's leg. There was an objection by the defendant. It was stated that the picture was taken by a competent physician who was familiar with the process. In commenting on the case the Court said:

"We might, if we so desired, rest our conclusions on the general character of the exceptions to this testimony. But we desire to place it on the ground that, verified by this picture, it was altogether competent for the purpose for which it was offered. There is no good reason why a court should not avail itself of the advances of science. Maps and photographs are often used to enlighten the court and jury. It is not to be understood, however, that every photograph as taken by the cathode, or *x*-ray process, would be admissible. Its competency, to be first determined by the trial judge, depends upon the science, skill, experience, and intelligence of the party taking the picture and testifying with regard to it, and lacking these important qualifications it should not be admitted; and even then it is not conclusive as to matters of fact, but is to be weighed like other competent evidence."

In DeForge *vs.* N. Y., N. H., and H. R. R., Supreme Judicial

[1] Smith *vs.* Grant, *Chicago Legal News*, 1896, vol. xxix., p. 145.
[2] *Southwestern Reporter*, June 16, 1897, vol. xlix., p. 445.

Court of Massachusetts,[1] the Court reversed a decision because the trial judge did not admit as evidence an *x*-ray negative and two pictures made from it. This case has little bearing upon the general admissibility of radiographs, as the exhibit which was offered on the part of the defendant was to rebut two *x*-ray pictures which had been exhibited on behalf of the plaintiff. The conclusion that was drawn from the pictures that were introduced was that there was a fracture of the foot. The defendants wished to show by their pictures that the plate from which the plaintiff's pictures had been made showed the appearance upon the uninjured foot. The defendants were not allowed to introduce their skiagraphs as evidence.

In commenting upon the exclusion of these pictures at the trial the Court said: "While a picture produced by an *x*-ray cannot be verified as a true representation of the subject in the same way that a picture made by a camera can be, yet it should be admitted if properly taken."

In Miller *vs.* Damon, Supreme Court of Washington, April 20, 1901,[2] in the progress of a trial a physician was examined as a witness and was permitted, over the appellant's objection, to exhibit to the jury an *x*-ray negative and to testify, based on the *x*-ray examination, that the leg had been fractured just below the knee-joint. The negative was permitted to go to the jury, which is assigned as error, it being argued that the witness should not have been allowed to give an opinion that the leg was fractured, but should have been confined to indicating on the skiagraph the particular appearances that were thought to determine a fracture and leave it to the jury to determine whether or not the appearances were there.

This argument was held to be faulty, as the physician was testifying as an expert who was not only familiar with fractures, but with their recognition by the *x*-ray. The method of the examination did not affect the competency of his testimony.

The introduction of the negative was not objectionable, as there is no good reason for making a distinction between a common photograph and one produced by the *x*-ray—that is, it is admissible as evidence when verified by proof that it is a true representation of an object which is the subject of inquiry.

In these four decisions there is practical unanimity of opinion as to the admissibility of skiagraphs, but there is a disposition to restrict their introduction only to those cases in which it can be shown that the radiograph has been taken by one skilled in the method. The further restriction that it is only in the nature of secondary evidence, like a map or drawing, that can be used to illustrate the testimony of experts, is undoubtedly the safer ruling. The danger is that a jury will regard a radiograph as substantially a photograph, when, in fact, it is only the picture of a shadow. With this distinction clearly explained, there is no good reason why they should not be introduced in evidence.

The greatest liability to error is in the interpretation of shadows of

[1] *Northeastern Reporter*, February 28, 1901, vol. lix., p. 669.
[2] *Pacific Reporter*, vol. lxiv., p. 804.

the carpus and tarsus. In one case a patient obtained damages for a fractured astragalus when there was an abnormal os intermedium. The mistake was discovered only by skiagraphing the sound foot, which showed the same deformity. Radiographs taken in one plane will sometimes fail to show a fracture when it is distinctly brought out by rays passed in a plane at right angles to the first exposure. Radiographs of the pelvis and of the skull are especially difficult of interpretation.

So far as we are aware, no case has reached the courts in which a failure to use the Röntgen rays has been alleged as negligence in a suit against a physician. The great value of the ray in diagnosis, the certainty with which some conditions may be recognized and corrected, and the precision which is obtained by its use in the adjustment of fractures may well lead to an allegation that failure to use them is negligence, particularly when their value is stated in the literature of the profession.

Numerous malpractice suits were brought because of the production of the x-ray burns when the rays were first used in medicine. So far as we are aware, none of them has been sustained, as it was clearly impossible that a physician could have had, when the x-rays were first used, any knowledge of the possibility of producing an x-ray burn or of the means of preventing it. Had these cases been sustained, it would in effect have altered the rule in so far that the physician would have been held to have assumed the risk of an investigation with the x-ray, and not the patient.

With a fuller appreciation of the dangers of the x-ray and an increasing knowledge of the mechanism by which burns are produced, they have greatly lessened in frequency. With increased perfection of apparatus and a lessened time required for the taking of the radiograph, the time of exposure to the x-ray has been shortened, and hence the danger of bad burns has been lessened. This improved technic ought in time practically to eliminate x-ray burns after the taking of a radiograph or the use of the fluoroscope. Carl Beck [1] has used it in diagnosis in 3000 cases, and has never noted irritation of the skin, and in only 2 cases was there epilation.

The comparatively short exposures with the latest and most approved methods may be still further shortened by the use of specially prepared paper and plates, by which results are reached comparable to instantaneous photography. With this highly sensitized paper sharp outlines of the diaphragm and heart have been obtained in one second.

Standardization of x-ray methods has been proposed, but so far no general agreement has been reached. We merely refer to it as a possibility of the future.

All the suits that have been instituted growing out of the x-ray have been due to burns and the consequent inflammation and gangrene which has resulted. By some writers it has been claimed that the term burn ought not to be applied to the changes produced by the x-ray. If this is true, then the word burn ought not be applied to the effects

[1] *Medical Record*, August 25, 1900, vol. lviii., No. 8, p. 281.

of the sun upon the skin. They are identical in most respects, being the effects of radiant energy. A sun-burn is not followed by immediate vesiculation, but an appreciable interval elapses before the erythema and the formation of blebs. The same is true of the *x*-ray burn, but commonly the time that elapses between the exposure and the development of the dermatitis is much longer, varying from a day or two to one or two weeks. A burn with the sun's rays is always superficial, while that from the *x*-ray may have a greater depth. The burns from the sun's rays may be exceedingly severe, and in some instances have caused death. Following the *x*-ray burns, there have been ulceration, gangrene, and ultimate exhaustion. Of course, if the area burned is large, the fatal progress of a case is accelerated.

As to the mechanism by which the burns are produced, there are various opinions. Some have regarded them as due to the electric radiation, while others attribute them to the direct action of the Röntgen rays. The latter is the only tenable position at this time, particularly if the experiments of Professor Thompson are considered, in which he excluded electric induction and, at the same time, produced over a limited area a Röntgen-ray dermatitis.

Robert Kiembock[1] has made an interesting study of the effect of the Röntgen rays upon the skin and the manner in which they cause inflammation and gangrene. He gives a table of the cases so far reported in which severe burning or sloughing of the skin was caused by the Röntgen rays. His chief contention is that the effect upon the skin is not due to electric induction. The arguments in favor of this seem conclusive. He shows that the character of the tube has something to do with causing the inflammation of the skin. A hard tube[2] gives rays that penetrate deeply, but do not furnish a marked contrast between the tissues. A tube of medium softness gives a good contrast and penetrates well, but, at the same time, has more effect upon the skin than does the hard tube. A very soft tube, with low voltage, gives abundant Röntgen rays, but they do not penetrate deeply, and, hence, do not give a good contrast ; the latter affect the skin deeply. The induction from the connections of the tube is quite as marked within as without the focus, and yet it is only there that the trophic effects on the skin are noted. In the periphery of the surface in which the Röntgen rays are brought in contact there is only a loss of hair ; as we pass toward the focus there is a progressive increase in inflammation, until, in the very center, a slough develops. Support of the argument that electric conduction has nothing to do with the development of inflammation of the skin is given by the fact that rubber gloves, which are non-conductors of electricity, are of no value in preventing the passage of the Röntgen rays, and are not of the least service in preventing inflammation of the skin.

[1] *Wien. klin. Wochenschr.*, December 13, 1900.

[2] By the terms "hard" and "soft" as applied to *x*-ray tubes is meant the degree to which the air is removed. A very soft tube is one that is imperfectly exhausted, and if the vacuum is too low, it will produce no *x*-rays. On the contrary, if the vacuum is too high, no *x*-rays will be produced. Between these two extremes all gradations exist, hence the terms hard and soft as applied to tubes are largely relative.

The cases reported by M. M. Sharpe[1] show the influence of the tube in causing dermatitis. The tube first used was excited by a 4-inch coil on a 100-volt alternating current with a 6-inch spark, later going up to a 7½-inch spark. The cases had all been under treatment for several weeks and were making good progress. The tube was then changed to one that gave less green rays and a visible stream of violet rays from the cathode. Within ten days everyone of the cases showed a dermatitis, undoubtedly due to the low vacuum of the tube. When the spark length was increased in this tube to 5.5 inches, and no violet rays were visible, there was no more dermatitis.

There has been much speculation regarding the pathology of x-ray burns. Rudis-Jacinski says that it is due to an acute, subacute, or chronic necrobiosis, due to an irritation of the peripheral sensory nerves. Reticular masses of delicate fibrous tissue have been found in the blood-vessels. Cases have been noted in which there was a peculiarly progressive course, the gangrene gradually spreading from the more superficial to the deeper tissues, and in some cases requiring an amputation. It is difficult in the reported cases to distinguish between what may be a primary effect of the Röntgen rays and that which is due to a secondary infection of the involved area. Unquestionably a destructive effect upon the cells, by which their vitality is seriously compromised, is the underlying condition. Wherever such a change occurs, extensive destruction may take place from secondary inflammation.

A certain idiosyncrasy or extraordinary susceptibility to the Röntgen rays has been noted; burns have been produced by exposures that, in the majority, cause no inflammation. These in future will be largely eliminated by the improved methods of taking radiographs and by the shortened exposure.

The symptoms of cutaneous disturbance are usually a slight erythema, which in mild cases resembles a sun-burn, and which commonly disappears in a few weeks. In others, in which the burn is more severe, there is some discomfort, burning, or itching. This is followed by an exfoliation of the epidermis. Usually the hair in the affected area falls. In burns of greater severity there is vesiculation, followed by a dry slough, in which there is but an imperfect line of demarcation between the healthy tissues and in which there is little tendency to separation. In a few instances pain of an atrocious character has been observed in the sloughing area. In some cases this symptom is not marked. Even after the slough separates, the ulcer left is indolent and very slow to heal.

We know of at least one case in which amputation of the foot was necessary on account of an intractable burn following an exposure of nearly two hours to the x-ray in the effort to obtain an accurate skiagraph. It was one of the early x-ray burns. When skiagraphs were first made, a number of cases in which the rays were applied to the head were reported in which severe burns were caused, and these may have contributed to the death of the patients; but in all cases there were complicating intracranial lesions.

[1] *Archives of the Röntgen Ray*, vol. v., No. 2, p. 50.

Maurice Rubel[1] reports a case in which death was apparently closely related to an x-ray burn of the abdomen. The patient, a woman forty-seven years of age, had pain in the left side and back, associated with nausea and chills, suggesting a stone in the pelvis of the kidney. A skiagraph was made, there being two exposures, each lasting twenty minutes. A Leeds' coil was used with a 3-inch spark, the tube being 16 inches from the plate and about 6 inches from the abdominal wall. Four days later a dermatitis appeared over an area half the size of the hand above and to the left of the umbilicus, accompanied by little papules and intense burning and itching.

Under external applications the local conditions improved for a time; then there was a relapse, the symptoms becoming more severe and the ulceration extending. An effort was made to heal the ulcer by excision and grafting. Prior to this operation there were attacks of severe constipation with nausea and vomiting. After the operation the abdominal conditions improved, but later the strength of the patient failed and she finally died.

No post-mortem examination was obtained, hence it is difficult to say that the abdominal disturbance was due to the x-rays. There have been examples in which the prolonged use of the x-ray on the abdomen has caused nausea and vomiting. It is conceivable that with the extensive injury to the external part in this case there may have been some damage of the abdominal contents.

Rubel[2] has found three other cases in the literature in which death was attributed to x-ray burns. All three of these cases, however, were complicated by conditions that might have contributed to the death, apart from the application of the Röntgen rays.

In view of our present somewhat imperfect knowledge of the Röntgen rays we feel that the following conclusions are justified:

1. The burns are produced by the direct action of the rays, being governed by the degree of vacuum of the tube, the nearness of the tubes to the parts, and time of exposure.
2. There is a marked idiosyncrasy on the part of some patients, but as yet there is no means of ascertaining those who are especially liable to the deleterious action of the rays.
3. Shortening the time of exposure is the best means of preventing an x-ray burn.
4. With our present available methods of rapid work no x-ray burn of any severity has so far been produced, though it is possible that even with these very short exposures people of extreme susceptibility might be affected.

The extended use of late of the x-rays in the treatment of varied conditions will unquestionably be followed by more or less burning, because the very purpose for which the rays are employed is to produce an inflammatory reaction in the tissues. They are now used extensively in the treatment of lupus, external cancers, and various skin affections.

[1] *Journal of the American Medical Association*, November 22, 1902, p. 1321.
[2] *Loc. cit.*

In addition, there is a well-directed effort toward treating pelvic cancers. It can easily be seen that, with the prolonged exposures necessary in the treatment of such conditions, severe burns may be produced. In view of the uncertainties, it would be well to warn patients of the risks which they run, and allow them to elect whether they will have the treatment or not. Certainly any statement that the *x*-rays are harmless, should the particular application be followed by a burn, might render the surgeon liable to an action. Already one suit has been brought as a result of the application of the *x*-rays in treatment.

LAWS RELATING TO THE INSANE.

A Digest of the Statutes of all the States and Territories, and of the District of Columbia, relating to the Commitment, Care, and Custody of the Insane.

STATUTES are arranged in the order of the States. The statutes of New York are given in full, as the care of the insane devolves wholly upon the State, which is the only State in the Union to adopt a uniform system of State care for its dependent insane, and to assume the entire financial cost thereof.

The official titles of the State and municipal institutions precede in each instance the statutes of a State. Lack of space forbids enumeration of the various private institutions for the insane.

A digest of the decisions relating to the powers and duties of committees of the person and estate of lunatics is also given at the end of this section.

ALABAMA.

(The Bryce Hospital, Tuscaloosa; The Mt. Vernon Hospital, Mt. Vernon.)

[References are to the official Code of Alabama.]

Definition.—The terms "lunatic," "insane," and "*non compos mentis*" include all persons of unsound mind. (Civil Code, § 1.)

Courts of Probate have, in the cases defined by law, original jurisdiction of—

6. The appointment and removal of guardians for minors and persons of unsound mind. (Idem, § 3363.)

Appointment of a Guardian.—The Court of Probate has power to appoint guardians for persons of unsound mind residing in the county having an estate, real or personal, and of such persons without the State having property within the county requiring the care of a guardian. (Idem, § 2255.)

Such guardian is not to be appointed until an inquisition has been had and taken. (Idem, § 2256.)

Inquisition Proceedings.—Upon the petition of any of the relatives or friends of any person alleged to be of unsound mind, setting forth the facts, and name, sex, age, and residence of such person, accompanied by an affidavit that the petitioner believes the facts therein stated to be true, the Court of Probate of the county in which such person alleged to be of unsound mind resides must appoint a day for a hearing thereon not more than ten days from the presentment of such petition. (Idem, § 2257.)

The judge must issue a writ directed to the sheriff, commanding him to summon twelve disinterested persons and to issue subpœnas for witnesses returnable at the time of trial. He must also direct the sheriff to take the person alleged to be of unsound mind, and, if consistent with health or safety, have him present at the place of trial. (Idem, § 2258.)

The jury must be impanelled and sworn. If any of the jurors from any cause do not serve, their places must be supplied from the bystanders. (Idem, § 2259.)

If the jury find the facts alleged in the petition to be true, and that such person is of unsound mind, the court must cause the petition and all the proceedings thereon to be recorded, and appoint a suitable guardian of such person. (Idem, § 2260.)

The alleged insane person must have notice of the inquisition; without such notice the proceedings are void. (McCurry *vs.* Cooper, 12 Ala., 823; Eslava *vs.* Lepetre, 21 Idem, 504; Molton *vs.* Henderson, 62 Idem, 426.)

If the person alleged to be of unsound mind is a resident of the county, and at the time of the application is confined in a hospital or asylum within or without the State, the inquisition may be had or taken without notice to him. (Civil Code, § 2261.)

Non-resident Persons of Unsound Mind.—The Court of Probate may appoint a guardian for a person of unsound mind having property within the State, if such person has been declared insane by a court having jurisdiction in the State of his residence. (Idem, § 2267.)

The application shall be in writing, verified, shall state the name, sex, age, and residence of such person, the court by which he was declared of unsound mind, and describe the property requiring the care of a guardian. A hearing must be given upon notice for three successive weeks by publication. (Idem, § 2268.)

Who may be Appointed Guardian.—The court must prefer in the appointment of a guardian the person who is of nearest relationship, and who will in the judgment of the court best manage the estate of the ward. (Idem, § 2269.)

The general guardian for the county must be appointed guardian of a person of unsound mind if no other suitable person applies for appointment and qualifies; and if there be no guardian, the sheriff must be appointed. (Idem, § 2270.)

Bond of the Guardian.—A guardian other than the general guardian for the county or the sheriff must enter into a bond with sufficient sureties payable to the judge of probate in a penalty to be prescribed by the judge, which must be not less than double the value of the personal property and the rental value of the real property for three years, with condition for the faithful performance of his duties. (Idem, § 2272.)

Land of a ward shall not be sold until the guardian has given a bond for double the supposed or estimated value of such land, payable to the judge of probate, and with condition that he will faithfully account for the proceeds of such sale. (Idem, § 2273.)

Revocation of Guardianship.—The insane person himself, or by next friend, may apply in writing to the Court of Probate for a revocation of the proceedings against him and of the letters of guardianship, the application to be accompanied by the certificate in writing of two physicians or of two other competent persons, stating that after examination of such person they believe him to be of sound mind. (Idem, § 2262.)

The court must appoint a day for the hearing thereof, not more than ten days thereafter, and the guardian and the person at whose instance the inquisition was had and taken must be cited to appear and show cause. If the guardian or such person appear and in writing deny the allegations of the application, the court must appoint a day for the trial of such contest, cause a jury to be summoned for the trial thereof, and the like proceedings

shall be had as upon the original inquisition. If there be no contest, a decree shall be entered revoking the proceedings of the inquisition and the guardianship. (Idem, §§ 2263–2266).

If at any time after his appointment the guardian becomes satisfied that the ward has been restored to sanity and is capable of managing his estate, and the judge of probate is of opinion from the proofs and the facts stated that such representation is correct, he shall make an order that the guardian be discharged and that the estate of the ward be restored. (Idem, § 2266.)

The Meaning of the Term Persons of Unsound Mind.—The term "Persons of unsound mind" includes idiots, lunatics, or the insane. (Idem, § 2271.)

Power and Duty of Guardians.—Within three months after his appointment the guardian must make an inventory of all the estate of his ward and return it upon oath to the court, which inventory must be filed and recorded. (Idem, § 2285.)

The guardian must manage the estate of his ward frugally and improve it to the best of his skill and ability. (Idem, § 2286.)

The guardian may publicly or privately lease the land of the ward for a term not exceeding one year, and make a report thereof to the Court of Probate. (Idem, § 2289.)

The Court of Probate may authorize the guardian to lease the land of the ward for a term not exceeding ten years, reserving rent payable annually; such lease being subject to revocation or disaffirmance by the ward upon restoration to sanity. (Idem, § 2290.)

The guardian must keep in good repair and condition the real estate of the ward, and he may make all such improvements and repairs thereon as are necessary and proper for that purpose; but such improvements or repairs shall not be made from the principal of the funds of the ward without an order of the Court of Probate. (Idem, § 2296.)

The Court of Probate may authorize a guardian to compromise any claim or debt due to the ward. (Idem, § 2301.)

The court may authorize the guardian to take real estate in compromise of any debt or claim. (Idem, § 2304.)

The court may authorize the guardian to sell debts or other choses in action of the ward which are of doubtful collection or recovery. This must be done upon the order of such court, granted upon the application in writing of the guardian verified by affidavit, but a report of the sale must be made to and confirmed by the court. (Idem, §§ 2308–2312.)

The court may authorize the guardian to sell any property, real or personal, when necessary for the payment of debts of such insane person incurred for the maintenance of such person or for the maintenance of his family. (Idem, §§ 2317, 2318.)

Notice of the sale must be given by advertisement for three successive weeks in a newspaper published in the county. (Idem, § 2326.)

A report of such sale must be made to and confirmed by the court. (Idem, § 2330.)

The court may set the sale aside (Idem, § 2331), vacate it and order a new sale (Idem, § 2332), or confirm the sale if it seems satisfactory (Idem, § 2334). The title of the ward is not divested until the purchase money is fully paid. (Idem, § 2334.)

The property of the ward may be sold for good cause shown, for the purpose of reinvestment. (Idem, § 2322.)

A guardian may invest the money of his ward in real estate, and if act-

ing in good faith shall not be individually responsible for a depreciation in the value of the land purchased with the funds of the ward, if such depreciation results from causes which cannot be prevented by the guardian. (Idem, § 2305.)

Real estate purchased by the guardian with the funds of the ward must be conveyed to the ward, but managed and controlled by the guardian for the benefit of the ward. (Idem, § 2306.)

The guardian is liable to the ward for any loss sustained by the failure or defect of title of the land purchased. (Idem, § 2307.)

The Court of Probate, on the application of the guardian, shall direct what portion of the income or of the principal of the estate of the insane person may be appropriated to the support of his family. (Idem, § 2313.)

Settlement of Guardian.—The Court of Probate has jurisdiction of the settlement, partial or final, of the accounts of the guardian. (Idem, § 2338.)

The guardian shall at least once in three years file in the court an account of his guardianship, accompanied with the vouchers verified by affidavit. The court shall appoint a day for the settlement, of which notice shall be given for three successive weeks in the manner directed by the court. On the day of settlement the court shall proceed to examine the vouchers and audit and state the account. If any voucher or item be rejected, all costs accruing on the contest or examination thereof shall be taxed against the guardian personally. Upon the final settlement, the partial settlement shall be presumed to be correct. (Idem, §§ 2339–2343.)

On the death, resignation, or removal of the guardian, or on the restoration to sanity of the ward, or on his death, the final settlement of the guardianship shall be made. Upon such settlement the guardian shall file in the Court of Probate a full account of the guardianship, accompanied by the vouchers and verified by affidavit. The court shall appoint a day for the settlement, with ten days' notice to the ward if restored to sanity, or to his personal representatives if dead. On the day appointed, the court shall proceed to examine the vouchers and audit and state the account. If any voucher or item be rejected, all costs accruing on the contest or examination thereof shall be taxed against the guardian. The court shall thereupon render a decree declaring the amount due the ward, which shall be entered and recorded and the account and vouchers shall also be recorded. (Idem, §§ 2344–2348.)

Compensation of Guardian.—The guardian is entitled to a commission of two and one-half per cent. on his disbursements, and two and one-half per cent. on his receipts, and on final settlement an allowance shall be made for all actual expenses necessarily incurred by him. Upon the final settlement, the guardian, if he has not been guilty of fraud or gross negligence, shall be allowed a reasonable commission, not exceeding two and one-half per cent., on the value of all personal property surrendered to the ward or to his representatives. But if the value of such personal property or of such moneys exceed twenty thousand dollars, the commission on the excess shall not be more than one per cent. (Idem, §§ 2350, 2351.)

Resignation and Removal of Guardian.—A guardian may resign by writing subscribed by him, filed in the Court of Probate. Such resignation does not affect the liability of the guardian or his sureties. (Idem, § 2362.)

A guardian may be removed for removal from the State, wilful failure to file an inventory or wilful disobedience to an order of the court, drunken-

ness, imbecility of mind, continued sickness rendering him incapable, conviction of felony, waste of the ward's property, neglect of his affairs, or for any other good and sufficient cause. (Idem, § 2363.)

An application for removal may be made by the ward or by next friend in writing, verified by affidavit, which shall specify the grounds on which the application is made. The court shall appoint a day for the hearing, of which at least five days' notice must be given to the guardian. (Idem, § 2364.)

On the day appointed, the court may proceed to hear the evidence and pass upon the application. If determined against the ward, the next friend shall be taxed with the costs; if against the guardian, he shall be taxed with the costs. The Court of Probate may, without an application by the ward, for any of the causes specified upon notice, remove a guardian. (Idem, §§ 2365–2367.)

Hospital Trustees.—The hospitals are managed by a board of seven trustees, appointed by the governor. Three are to reside near the hospital and four in other parts of the State. Such trustees make by-laws for the management of the institutions under their charge. (Idem, §§ 2545, 2546.)

The superintendent of the hospital is appointed by the board. He shall be qualified to practise medicine under the laws of Alabama, and possess "prompt business habits, and of a humane, kindly disposition." The superintendent, with the concurrence of the board, appoints the assistant physicians, stewards, supervisors, and other employés. (Idem, § 2548.)

A person shall be considered "fit to be a patient in the hospital who exhibits, in the first place, such a degree of brain debility or mental aberration as to render him constantly troublesome, or a nuisance, or dangerous to others, or dangerous to his own life or person, or dangerous to property; in the second place, this disability must not be transient like delirium in a fever, but of more or less permanent character; in the third place, lack or loss of mental ability to properly conduct his usual work or business shall be considered along with aberrant conduct in determining the question of a person's insanity." (Idem, § 2549.)

In order of admission, recent cases of both classes may have preference over those of long standing. (Idem, § 2550.)

Commitment of Insane Persons.—When a person in indigent circumstances becomes insane, application shall be made by his friends, or any other person in his behalf, to the judge of the Probate Court in the county where he resides; and such judge shall without delay make application to the superintendent of the hospital for his admission, and accompany the application with full and satisfactory answers to certain questions relating to the condition of such person. If the patient can be received, the judge shall call one respectable physician and other trustworthy witness to investigate fully the facts in the case, and either with or without the verdict of a jury decide the case as to sanity and indigence. If the judge believes that satisfactory evidence has been advanced showing the patient to be insane, and that his estate is insufficient to support him and his family, the judge shall issue a certificate and send him within thirty days to the hospital, to be supported there at the expense of the State. (Idem, §§ 2551–2553.)

Discharge is made under the rules adopted by the board of trustees by the superintendent in the event of the recovery of a patient from the attack of insanity. The local board of trustees may discharge a patient from the hospital at any time. The superintendent of the hospital may grant a furlough for a period not exceeding six months to such of the harmless and

convalescent patients who may, in his opinion, be benefited by the change. The friends of such patients shall pay all travelling expenses to and from the hospital. Persons confined in such hospitals shall be entitled to the right of habeas corpus. (Idem, § 2560.)

ARIZONA.

(Territorial Insane Asylum, Phœnix.)

Insane Asylum; Directors.—The Territorial Insane Asylum of Arizona is located near the city of Phœnix. It is under the management of a board of three directors, nominated by the territorial governor and confirmed by the legislative council, and two of whom hold office for two years and one for four years. The directors are required to execute a bond in the sum of ten thousand dollars, conditioned for the faithful discharge of their duties. The directors have full power and authority to manage the affairs of the asylum. (Statutes of Arizona, §§ 2461, 2464.)

Officers.—The board of directors shall appoint a secretary and treasurer and a resident physician, who is the superintendent of the asylum. (Idem, §§ 2477, 2480.)

Superintendent.—The resident physician shall be a college graduate in medicine and surgery and have practised not less than four years. He is the superintendent of the asylum, and as such is the chief executive officer and has the general superintendence of all the affairs of the asylum, subject to the laws and regulations adopted by the board of directors. He is to ascertain daily the condition of the patients; and, with the approval of the board of directors, appoint assistants and attendants. He may discharge any of such assistants or attendants at his sole discretion, and shall record each discharge, with the reasons therefor, in one of the books of the asylum. He shall direct all the employés of the asylum and maintain discipline, and shall cause to be kept books containing a record of all the affairs of the asylum. (Idem, § 2481.)

By-laws and Regulations; Reports; Visitation; Meetings.—The board of directors are empowered to make by-laws and regulations for the government of the asylum. They shall make an annual report to the legislature, showing receipts and expenditures, the condition of the asylum, the number of patients admitted and the number remaining. They shall make a thorough monthly visitation. They shall meet monthly, and a majority constitutes a quorum. (Idem, § 2482.)

Estimates; Purchases.—The board of directors shall estimate monthly, in advance, the probable expenses of the asylum, and the auditor of the Territory shall draw his warrant for such amount in favor of the directors on the first day of each month, and the treasurer of the Territory shall pay the same. The superintendent shall make all purchases under the direction of the directors. (Idem, § 2483.)

Patients to be Received.—All persons adjudged insane by a court of competent jurisdiction shall be received. If estates of persons admitted are chargeable or friends or relatives are willing and able to pay, rates are to be fixed by the board, and collected and paid into the treasury of the territory to the credit of the insane asylum fund. (Idem, § 2484.)

Determination of Insanity; Confinement.—Any probate judge has power to hear and determine questions of insanity. Application is to be made upon oath setting forth that a person by reason of insanity is dangerous and is at large. The alleged insane person shall be brought before the

judge; two or more witnesses acquainted with the person shall be summoned, who shall testify as to the conversation, manners, and general conduct of the alleged insane person. One or more graduates in medicine and reputable practitioners thereof shall be present at the examination, and after hearing the facts detailed by the other witnesses and a personal examination of the alleged insane person, shall upon oath state: (1) His judgment as to the insanity of the person; (2) whether it be dangerous to the person of the alleged lunatic or to the community; (3) whether the insanity is permanent or temporary. If upon such proof and statements the judge is satisfied that the person is insane, he shall by order direct the confinement of such person in the Territorial Insane Asylum. The judge upon such examination is to make inquiry into the ability of the insane person to bear the expenses and cost of commitment and confinement in the asylum. If such person have property, a guardian is to be appointed, whose duty it is to receive such property and pay therefrom the costs of the commitment and support of such insane person. The costs of commitment and transportation of all indigent insane are to be paid by the county. (Idem, §§ 2156, 2160.)

ARKANSAS.

(State Asylum, Little Rock.)

[References are to Sandels and Hill's Digest of Statutes.]

Probate courts possess the superintending control over guardians having the care, custody, and management of idiots, lunatics, habitual drunkards, and persons of unsound mind, and may provide for the safekeeping of such persons, and the maintenance of themselves and their families and the education of their children. (Statutes of Arkansas, § 3814.)

Inquisition.—Upon information in writing that any person in the county is an idiot, lunatic, or of unsound mind, if satisfied that there is good cause for the exercise of its jurisdiction, the Probate Court shall cause the person to be brought before such court and inquire into the facts by jury. If any sheriff, coroner, or constable discovers such a person in the county, he shall make application to the court for the exercise of its jurisdiction. If found by the jury that the person is of unsound mind, the court shall appoint a guardian of the person and estate of such insane person. The costs of the proceedings shall be a charge on his estate, or, if that is insufficient, upon the county. If the person alleged to be insane is discharged, the costs are paid by the person instituting the proceedings, unless such person is an officer, in which case the costs shall be paid by the county. (Idem, §§ 3815–3820.)

Guardians, their Powers and Duties.—Before entering upon his duties the guardian shall execute a bond for the faithful performance of such duties and the judicious management of the estate of his ward. Additional bonds may be required, in the discretion of the court. The guardian shall take charge of the person committed to his charge and provide for his support and maintenance. He shall take into his possession the goods, chattels, moneys, and all evidences of debt and of writing touching the estate of the person under his guardianship. Within three months he shall make out and file in the office of the clerk of the court an inventory of the estate of his ward. Additional inventories shall be filed whenever other property belonging to the estate may be discovered. The court may make orders for the restraint, support, and safekeeping of such person; for the maintenance of his estate and the support and maintenance

of his family and education of his children, out of the proceeds of his estate. (Idem, §§ 3821-3831.)

Sale, Mortgage, and Lease of Real Estate.—If the personal estate be insufficient for the discharge of the debts and maintenance of the family, or education of the children of the insane ward, the guardian shall petition the court for authority to mortgage, lease, or sell such part of the real estate as is necessary to supply the deficiency. The courts shall direct the time and terms of the sale, mortgage, or lease, and the manner in which the proceeds shall be secured and the income applied. The court shall direct the manner of making the sale, which shall be at public vendue to the highest bidder, and the guardian shall report the proceedings to the court. If the court approve the proceedings, the guardian shall execute a deed to the purchaser. If the report be disapproved, the court may set aside the sale and order the refunding of all money paid. When the court shall order a lease or mortgage, no deed or instrument shall be executed until approved by the court. (Idem, §§ 3832-3841.)

When Ward may be Supported by County.—If the estate of any ward is insufficient to maintain himself and family, or educate his children, his guardian may apply to the County Court for an appropriation from the county treasury for the support of his ward. The petition shall be accompanied by an account of his guardianship, an inventory of the estate of the ward, and a list of the debts due from such ward. If the court is satisfied that the estate is insufficient for such purposes, it may order such sum to be paid to the guardian out of the county treasury as is sufficient to provide for the support of his ward. (Idem, §§ 3848-3851.)

Restoration of Insane Person.—If any person allege in writing that the person declared of unsound mind is restored to his right mind, or to correct and sober habits, the court shall cause the facts to be inquired into, and if it be found that such person has been restored, he shall be discharged from care and custody and his property shall be restored to him. (Idem, §§ 3852, 3853.)

Settlement of Accounts of Guardian.—All accounts of guardians shall be settled on the termination of the guardianship. The guardianship is terminated by the restoration of the ward, his death, or removal from office. (Idem, §§ 3854, 3855.)

Confinement of Insane Person by Guardian.—If a person be furiously mad, or so far disordered in his mind as to endanger his own person or the person or property of others, it shall be the duty of the guardian, or the person under whose care he may be, and who is bound to provide for his support, to confine him in some suitable place until the Probate Court shall make an order for his proper restraint, support, and safekeeping. If such person is not confined, any judge of a court of record, or any two justices of the peace, may cause such insane person to be apprehended, and may provide for his confinement in some suitable place until the court shall make further order thereon. (Idem, §§ 3858, 3859.)

Insane Persons found at Large.—Insane persons at large and not in the care of some discreet person, shall be arrested by any peace officer and taken before a magistrate. Such magistrate shall make such orders as may be necessary to keep him in restraint until he can be sent to the lunatic asylum. He may be confined in the county jail if there are no persons to whose custody or care he may be committed. (Idem, §§ 3861, 3862.)

Insane Paupers.—When there is a poor home in the county, all insane

persons shall be taken care of as other paupers, and the County Court may make such additional compensation for taking care of insane paupers as may be deemed just. (Idem, § 3863.)

Lunatic Asylum.—The Arkansas State Asylum is located at Little Rock. It is governed by the Board of Trustees of Charitable Institutions, which also has charge of the school for the blind and the deaf mute institute. It is composed of six members appointed by the governor, one from each congressional district. (Idem, § 3928.)

The trustees shall appoint a superintendent, who shall be a skilled physician, and who shall hold his office for a term of four years. They shall maintain an effectual inspection of the asylum, for which purpose one or more of them shall visit it at least once in every week, and the whole board once in three months. (Idem, §§ 3977–3980.)

The Superintendent.—The superintendent appoints all subordinate officers, and is the general superintendent of the buildings, grounds, and farm, with all their fixtures and stock; and has the direction and control of all persons therein, subject to the by-laws and regulations of the trustees. He is directed to ascertain daily the condition of the patients and prescribe their treatment. (Idem, § 3984.)

Admission to Asylum.—Any person may be admitted to the asylum as a patient who is a citizen of the State, and who may be insane. Any reputable citizen may file a written statement with the county and probate judge, that to the best of his belief the alleged insane person ought to be admitted, and that his being at large is dangerous to the community or prejudicial to his chances to recover. Thereupon a time is appointed by the judge for a hearing, and at such time such competent witnesses as are produced are heard. In addition to the testimony of such witnesses the judge shall cause the alleged insane person to be examined by two reputable, competent, and disinterested physicians at different times and places separately, who shall present in writing the result of the examination, including the answers to certain questions which are prescribed by statute. If satisfied that such person is insane, he shall so decide, and without delay transmit to the superintendent of the asylum his decision in writing, with copies of the statements taken in the proceeding. The superintendent shall notify the judge of his readiness to admit the insane person into such asylum if there be room unoccupied. If there be no room unoccupied, he shall notify the judge of the fact and return the papers indorsed accordingly. On receipt of notice of the superintendent's willingness to receive the patient he shall be transferred to such asylum by the sheriff or any person deputized by the county judge. Such insane person shall be supported in such asylum at public expense unless such person have more than sufficient estate for the support of his family. (Idem, §§ 3990–3999, as amended by Laws of 1899, Act 74.)

Criminal Insane.—All persons acquitted of a criminal offence on the plea of insanity shall be received in the asylum, and there detained until restored to reason. No person becoming insane while in confinement in a penal institution shall be received into the asylum, but shall be treated for the disease within the penitentiary. (Idem, §§ 4001–4005.)

Classification of Insane.—Insane persons committed to the asylum are classified as "acute," "chronic," and "probably incurable," such classification to be determined by the duration of the disease and such complications as are known to render recovery doubtful, if not impossible. Cases of one year's duration are "acute"; of over one year's duration, "chronic"; and

cases complicated with epilepsy, imbecility, or feeble-mindedness, deformities of skull from injuries, old age, or general paralysis, are "probably incurable." (Idem, § 4006, as amended by Laws of 1897, Act 36.)

CALIFORNIA.

(Stockton State Hospital, Stockton; Napa State Hospital, Napa; Agnews State Hospital, Agnews; Southern California State Hospital, Patton; Mendocino State Hospital, Mendocino.)

[References are to official Codes of California.]

Appointment of Guardians of Insane and Other Incompetent Persons.—Power of appointment is vested in the Superior Court. Appointment is made upon petition of relative or friend that such person is insane or for any cause incompetent to manage his property. Notice must be given such person not less than five days before hearing. If it appears upon the hearing that the person is incapable of taking care of himself and managing his property, a guardian shall be appointed. (Cal. Code Civ. Pro., §§ 1763, 1764.)

Proceedings to Determine Restoration of Insane.—An insane person, or any relative or friend, may apply by petition to court to have the fact of his restoration judicially determined. The court shall appoint a day for hearing, and upon request order investigation before a jury. Notice of the trial shall be given to the guardian of the person, to his or her husband or wife, or to his or her father or mother, if living in the county. The guardian or relative of such person may contest the right to the relief demanded. (Idem, § 1766.)

Powers and Duties of Guardians.—The guardian has the care and custody of the person of his ward and the management of all his estate. (Idem, § 1765.)

The guardian shall pay all the ward's debts out of his personal estate or income of his real estate, if sufficient; if not, then out of real estate upon obtaining order for sale. He shall settle all accounts of the ward, demand, sue for, and receive all debts due him. He shall manage the estate of his ward frugally and without waste, and apply the income for suitable maintenance of ward and his family; and if insufficient, may sell the real estate upon order of the court and apply the proceeds of such sale to such maintenance. The guardian shall be allowed credits for proper advances for the benefit of the ward upon proper vouchers, etc. The court may direct the guardian to pay the persons supplying the ward with suitable maintenance, etc. The guardian may join in and assent to a partition of the real estate of the ward. The guardian shall return to the court an inventory of the estate of his ward within three months after his appointment, and annually thereafter. Inventories shall be recorded by the clerk of the court. The guardian shall upon the expiration of a year from the time of his appointment, and as often as required, present to the court his account for a settlement. Every guardian shall be allowed the amount of his reasonable expenses, and also such compensation as the court deems just and reasonable. (Idem, §§ 1768–1776.)

Sale of Property of Ward and Disposition of Proceeds.—When income of the estate is insufficient to maintain the ward and his family, the guardian may sell real or personal estate upon order of the court. The court may order sale of the property of the ward for the purpose of reinvestment. The proceeds of the sale shall be applied to the purposes for

which it was made, and the residue invested until the capital is required for such purposes. If sold for purposes of investment, investment shall be made according to the guardian's best judgment or in pursuance of an order of the court. The order shall be made upon petition containing a statement of facts and circumstances showing the necessity for such sale. A copy of the order shall be served on the next of kin and all persons interested in the estate at least fourteen days before a hearing of the petition. The order of the court shall specify the causes of the sale. A guardian authorized to sell real estate shall give a bond to be approved by the court. Such order shall not continue in force longer than one year after being granted. (Idem, §§ 1777–1792.)

General Provisions.—The court may cite persons suspected of having concealed, embezzled, or conveyed any of the property belonging to the ward or to his estate, upon complaint of the guardian, ward, creditor, or other person interested in the estate, and may summon him in the same manner as persons suspected of concealing the effects of a decedent. (Idem, § 1800.)

A guardian may be removed if incapable of discharging his trust, or if he has wasted or mismanaged the estate, or failed for thirty days to render an account as required by the court. A guardian may resign when it appears proper to allow the same. (Idem, § 1801.)

Guardianship is terminated when it appears that it is no longer necessary. (Idem, § 1802.)

The court may require a new bond by the guardian when deemed necessary. Every bond of a guardian must be filed in the office of the Superior Court of the county. No action can be maintained on such bond unless commenced within three years after the discontinuance of the guardianship. No action can be maintained for the recovery of any estate sold by a guardian unless commenced within three years from the termination of the guardianship. (Idem, §§ 1803–1805.)

Custody of Insane Persons.—Any person of unsound mind may be placed in an asylum upon the order of the Superior Court of the county in which he resides as follows: 1. The court shall be satisfied upon examination in open court and in the presence of the person, from the testimony of two reputable physicians, that such person is of unsound mind and unfit to be at large. 2. After the order is granted the person alleged to be insane, his or her husband or wife, or relative to the third degree, or any citizen, may demand an investigation before a jury, which shall be conducted in all respects as under an inquisition of lunacy. (Idem, § 258.)

State Commission in Lunacy.—The State Commission in Lunacy consists of five members, three of whom are the State Board of Examiners (*i. e.*, the governor, secretary of State, and attorney-general), the general superintendent of State hospitals, and the secretary of the State Board of Health. The general superintendent shall be a reputable physician, a graduate of a medical college, and have had six years' experience in the care and treatment of the insane. His salary is $4000 per year; he receives also his necessary travelling expenses. The other members of the commission serve without compensation. The general superintendent is appointed by the governor. The commission is charged with the execution of the laws relating to the insane, and is required to examine all institutions authorized by law to receive and care for the insane. (Statutes of 1897, ch. 227, art. 1, §§ 3–5.)

Powers of General Superintendent and Commission.—The general

superintendent is required to visit all institutions at least twice in each year. The records and methods of each institution are to be examined by the commission, and recommendations as to such methods not inconsistent with the school of medicine in charge of such institution may be made. By-laws are required to be uniform for each hospital. (Idem, §§ 7, 8.)

Examiner in Lunacy.—Certificates of examiners in lunacy are issued by a judge of any Superior Court in the form prescribed by the commission, showing that the examiner is a graduate of an incorporated medical college, and has been in the actual practice of his profession for at least five years. Such certificates are filed with the county clerk, and a certified copy thereof forwarded to the commission. No examiner is qualified until he has received from the commission an acknowledgment of the receipt of such certificate. (Idem, art. 1, § 9, and art. 3, § 2.)

State Hospitals.—Each State hospital is under the control, subject to the statutory powers of the commission, of a board of five managers, appointed by the governor. The managers appoint the medical superintendent and treasurer. The medical superintendent appoints assistant physicians and other subordinates, officers, and employés. (Idem, art. 3, §§ 1, 2, 4–6.)

Commitment.—Application for commitment to a State hospital may be made by a peace officer or the chief officer of any city, city and county, county, or receiving hospital, or by any relative or friend of the alleged insane person. Notice shall be served on the relatives or friends of the alleged insane person in case the application is not made by them. The application shall be accompanied by certificates of lunacy made by at least two examiners in lunacy. The court directs a hearing on the demand of a relative or near friend. If he is satisfied that the person examined is insane, an order of commitment is made by him. A trial by jury may be demanded by the person asking for the hearing, if he is dissatisfied with the decision of the judge. The poor and indigent insane are supported as wards of the State. If the relatives or friends desire homeopathic treatment for the insane person, he is committed to the Southern California State Hospital. (Idem, art. 3.)

COLORADO.

(Colorado State Insane Asylum, Pueblo.)

The terms "insane person" shall be construed to include every idiot, *non compos*, lunatic, or distracted person. (Mills' Colorado Statutes, 118, § 4, 185.)

The term lunatic shall be construed to include idiots, insane and distracted persons, and every person who, by reason of intemperance or any disorder or unsoundness of mind, shall be incapable of managing and caring for his own estate. (Idem, ch. 80, § 2968.)

Appointment of a Conservator.—The County Court may appoint a fit person to be a conservator of the estate of a lunatic upon complaint of any reputable person that a person is a lunatic or insane person, that he has property and is incapable of properly managing the same. The court shall order a jury of six jurors to be summoned to ascertain whether such person is so incompetent of managing his affairs. (Idem, § 2935, as amended by Laws of 1891, pp. 331, 332.)

Bond; Powers and Duties.—A conservator shall give his bond with sufficient sureties conditioned for the faithful discharge of his duties. He shall have the entire charge of the estate of such lunatic, and shall make an inventory of such estate in the same manner as administrators are required

to inventory the estates of their intestates. He may collect and take possession of all the property of the lunatic. He may collect all debts due such lunatic and maintain necessary actions at law. (Idem, §§ 2936–2938.)

The conservator shall apply the annual income of the estate and proceeds of all sales to the payment of the debts of such lunatic, and the surplus to the maintenance of such lunatic and his or her family, and the education of the children. (Idem, §§ 2939, 2940.)

Settlement of Claims.—The County Court may allow demands against the lunatic to be satisfied out of the estate upon notice to the conservator. All claims must be presented within one year. All judgments are enforced against the estate in the manner provided for the collection of judgments against executors and administrators. The wife of a lunatic may select and take the articles which would be allowed to her as his widow in case of his death; but if the lunatic is solvent, the court may make further allowance to the wife for her support and the support and education of any minor children. (Idem, §§ 2941–2944.)

Sale of Property.—A conservator may sell personal property of a lunatic and lease lands as directed by the County Court upon an order; no lease shall be for a term exceeding five years. The order for the sale shall specify the amount thereof which shall be sold. Property exempt from execution shall not be sold except for the support of the lunatic or his family. Real property may be sold or mortgaged for payment of debts and support of the family, upon petition of the conservator, upon the order of the court granting the same. (Idem, §§ 2945–2947.)

Account of Conservator.—The conservator shall present to the court an account of his administration of the affairs of the lunatic six months after his appointment, and with every alternate term thereafter, setting forth the amount of money received and the sources from which received, the amount of personal property and choses in action on hand, and the several sums of money expended by him, to be accompanied by vouchers. The County Court may apportion such money among the creditors of the lunatic. The surplus shall be paid by the conservator to the wife. (Idem, §§ 2949–2952.)

Conservators shall receive reasonable compensation at the discretion of the court for their services. A conservator may be removed for any default or misconduct in office. He may resign upon making final and true account of his administration of the affairs of the lunatic. Such resignation is not to be construed to discharge the conservator or his sureties from any liability. Upon resignation he shall deliver to his successor all property, etc. (Idem, §§ 2953–2956.)

Confinement and Support of Lunatics.—If a lunatic have no relatives or friend who will care for him, the County Court shall order him to be placed in the State Insane Asylum, or other hospital or place suitable for the treatment of the insane. If such lunatic shall have an estate in the hands of his conservator, the conservator shall pay the sum expended for the support of the lunatic out of such moneys. If such lunatic has no estate, the amount so expended shall be paid by the county commissioners, except that if confined in the State Asylum no charge is made. (Idem, § 2957, as amended by Laws of 1893, p. 332.)

Restoration of Lunatic.—If such lunatic shall be restored to reason, all his property in the hands of the conservator shall upon the order of the court be restored to him. In case of the death of the lunatic the property shall be surrendered by the conservator to the heirs, executors, or administrators. (Idem, § 2959.)

Discharge of Insane Persons.—An inquest may be had in the County Court on information in writing presented by any person setting forth that the insane person has been restored to reason. If upon such inquest it shall be found that such person has been restored, he shall be immediately set at liberty. If confined in a State insane hospital, the superintendent shall discharge him and transmit to the County Court committing such patient a notice that he has been mentally restored and discharged. Upon the discharge of any lunatic, if there has been appointed a conservator, the court shall require the conservator to appear and settle his accounts. The expenses attending any inquest shall be paid out of the estate of the lunatic upon the order of the County Court. If there be no estate, such expenses shall be paid by the county, which shall be reimbursed by the State. (Idem, § 2961, as amended by Laws of 1893, pp. 332, 333.)

Arrest and Commitment of Lunatics.—Whenever any reputable person shall file with the County Court a complaint duly verified, alleging that any person is insane, or distracted so as to endanger himself or his property, or others and their property, the judge shall issue an order directed to any sheriff for the apprehension of such alleged insane person. When any sheriff or constable shall find within his county any insane person at large, he shall apprehend such insane person without an order of the court; and when so arrested, either with or without an order of the court, he shall be taken forthwith before the county court, or a judge thereof, and an inquest shall be held without delay; and until the determination of such inquest such alleged insane person shall be confined in a hospital or other suitable place to be designated by the County Court or by a judge thereof. If upon such inquest it be found that such alleged insane person is insane so as to endanger himself or others, it shall be the duty of the court to order that the patient be immediately transferred to the State Insane Asylum. The court may, in its discretion, designate some hospital or other suitable place where such patient shall be confined and treated with all the skill possible. (Idem, § 2962, as amended by Laws of 1893, p. 333.)

Insane Person not to be Confined in Jail.—No insane person shall be confined in a jail or lockup unless he is violent and his safety absolutely demands such confinement; and under no conditions shall he be confined in such jail or lockup for longer than ten days. (Colorado Laws, 1893, p. 335.)

Physician Examiner in Lunacy.—No physician shall testify to the insanity of a person for the purpose of securing his commitment unless such physician is of reputable character, a graduate of a medical college, a permanent resident of the State, and in the actual practice of his profession. Such qualifications shall be certified to by a judge of a court of record, and the certificate filed with the clerk of the County Court. (Idem, p. 336, § 5.)

State Asylum.—The State Asylum is managed by a State Board of Lunacy Commissioners appointed by the governor. The superintendent is appointed by the board, and shall be a physician, a graduate of a medical college, of ten years' professional experience and five years' experience in a hospital for the insane. Insane criminals are confined therein in a separate ward. The buildings hereafter erected are to be on the "cottage plan," each building to accommodate not less than fifty nor more than one hundred patients. (Laws of 1893, p. 337, §§ 9, 100, and Laws of 1899, p. 257.)

CONNECTICUT.

(Connecticut Hospital for the Insane, Middletown, Conn.)

Care and custody of insane persons and their property are vested in the Probate Courts.

Conservators.—A Court of Probate may appoint a conservator, on application of relatives or selectmen, of persons incapable of managing their affairs, and who by waste of their estates are likely to be reduced to want. Upon such application a citation shall issue to the alleged incompetent person and to relatives. (Connecticut General Statutes, ch. 45, §§ 475, 476. III.–36.)

Duties of Conservators.—A conservator is required to return an inventory of the estate of the incapable person, to manage his estate, to apply the income thereof, or principal if necessary, to his support and to the support of his family, and to pay his debts, and may sue for and collect all debts due to him. A Court of Probate may order the sale of real estate of a ward, and the conservator shall invest the avails of the estate, if not required for the immediate support of the insane person or the payment of his debts, either in other real estate or in such manner as trust funds may be legally invested. (Idem, §§ 478–480.)

Restoration of Estate of Ward.—If a Court of Probate shall find a ward restored to his mental capacity, it shall order what remains of his estate to be returned to him; and if such ward shall die, the property in the hands of the conservator shall be turned over to the ward's administrator or executor. (Idem, § 481.)

Effect of Removal of Ward to Another Probate District.—If a ward removes into another probate district, the Probate Court of such district may, upon application of the proper parties, appoint some person resident in such district as his conservator. The former conservator shall settle his accounts with the Court of Probate, and transmit to the new conservator all the personal property and other effects in his possession, and shall thereafter cease to be such conservator, and shall be succeeded in all his rights by the new conservator. (Idem, § 482.)

Confinement of Insane.—No person shall be committed or admitted to an asylum without an order signed by a judge of probate, as hereinafter provided.

Whenever any person in this State shall be insane, or supposed to be insane, any person may make complaint in writing to any judge of probate within whose district the person complained of shall reside, alleging that such person is insane and is a fit subject to be confined in an asylum; but when any insane person who ought to be confined shall go at large in any town, any person may, and the selectmen thereof shall, make a like complaint to the judge of probate within whose district such town is included. After receiving such complaint the judge to whom it is made shall forthwith appoint a time, not later than ten days after receipt of said complaint, and a place within said district, for a hearing upon said complaint, and shall cause reasonable notice thereof to be given to said complainant, to the person complained of, and to such relative or relatives of said person or to any person interested in said person, as said judge shall deem proper, and may adjourn said hearing from time to time for cause. Said judge may issue a warrant for the apprehension and bringing before him of said person complained of, and shall see and examine said person if in his judgment the condition or conduct of such person renders it necessary and proper so to do;

or state in his final order why it was not deemed necessary or advisable so to do. (Idem, 1889, ch. 162, §§ 3, 4.)

A certificate signed by two physicians, neither of whom is connected with any asylum nor related to the person complained of, shall be filed with such judge. Each must have personally examined the person within five days of signing the certificate. A copy of said certificate shall be attached to the final order of said judge and delivered with the order to the superintendent of the asylum. (Idem, § 5.)

If on hearing the judge shall find the person insane, and that he ought to be confined, he shall make an order in writing directing some proper officer to convey said insane person to the asylum named in the order. (Idem, § 6.)

All insane persons confined in any asylum shall be entitled to the benefits of the writ of habeas corpus, and the question of insanity shall again be determined by the judge issuing the writ. Said writ may be applied for by the insane person, or on his behalf by any relative or friend or person interested in his welfare. (Idem, § 16.)

An attorney-at-law regularly retained by or on behalf of any patient in an asylum, or any medical practitioner properly designated, may be admitted to visit the patient at all reasonable hours if, in the opinion of the superintendent of the asylum, such visit would not be injurious, or if a judge of the Superior Court first orders in writing that such visit be allowed. Insane persons detained in an asylum are to be furnished with materials for communicating with any suitable person. (Idem, §§ 19, 20.)

All asylums are subject to visitation of the State Board of Charities, and shall be so visited and inspected once in six months in each year. (Idem, § 22.)

Persons wilfully conspiring with any other person to commit to an asylum any person who is not insane, or any person falsely certifying to the insanity of any person, and any person falsely reporting that a person is insane, shall be punished by a fine of not more than one thousand dollars or by imprisonment in a State prison for not longer than five years, or by both. (Idem, § 23.)

License of Private Institutions.—No institution for the treatment or detention of insane persons shall be conducted or maintained without a license granted by the governor. Application shall be made under oath to the governor, stating the proposed location, the number of persons for whom accommodation will be provided, the name of the person to be placed in charge, and the previous experience which such person has had in the care and treatment of insane persons. Every private institution must be in charge of a physician of at least three years' experience as a medical attendant in an institution for the insane. The license is revokable for improper management or violation of law, after an opportunity for the licensee to be heard. (Idem, 1897, ch. 215.)

Insane Paupers and Indigent Persons.—If a pauper is insane, the selectmen are required to apply to a Court of Probate for his commitment. If an indigent person is insane, application may be made by any person in his behalf. Two physicians are appointed by the court to examine the alleged insane person. If found insane, he is committed to the State Hospital. If a pauper, the town where he resides pays two dollars per week for his support, and the State the rest. If an indigent person, the person making the application pays two dollars per week, and the State the remainder. (Idem, 1895, ch. 180.)

DISTRICT OF COLUMBIA.

(Government Hospital for the Insane, Washington.)

The hospital provides for the insane of the army and navy of the United States, and of the District of Columbia. (Revised Statutes of United States, § 4838.)

Executive.—The chief executive officer is the superintendent, who is appointed by the Secretary of the Interior, and who shall be a resident physician and who shall devote his entire time to the work of the institution. (Idem, § 4839.)

Nine citizens appointed by the President constitute a Board of Visitors, which makes the by-laws governing the institution, examines, makes recommendations for, and reports on the same annually to the Secretary of the Interior. (Idem, § 4840.)

Patients.—The superintendent, upon the order of the Secretary of War, the Secretary of the Navy, and of the Secretary of the Treasury, respectively, shall receive and keep in custody until they are cured or removed by the same authority which ordered their reception, insane persons belonging to the army, navy, marine corps, and revenue cutter service; civilians employed in the quartermaster and subsistence departments of the army who may become insane in such employment; indigent persons who become insane in such service, or who become insane within three years after their discharge therefrom; insane persons resident in the District. (Idem, § 4843.)

Commitment.—Patients are committed on the certificate of the Secretary of the Interior, who in turn receives a certificate from a judge of the Supreme Court of the District. The judge's certificate is made after examination by two physicians, and on affidavits of two householders as to indigency, and annexed thereto are the affidavits of such physicians. (Idem, §§ 4845, 4846.)

Expenses.—Patients other than indigent ones pay a sum not to exceed $4.50 per week. The expense of the institution falls half on the government and half on the District. A patient may be discharged upon bonds being given. (Idem, 1 supp., § 289.)

FLORIDA.

(Asylum for the Indigent Insane, Chattahoochee.)

The terms "insane person" and "lunatic" include every idiot, non compos. lunatic, and insane person. (Revised Statutes of Florida, § 1.)

Lunatic Asylum.—The asylum for indigent insane is located at Chattahoochee, and is controlled by the Board of Commissioners of State Institutions. Such board appoints and removes at pleasure the superintendent, and employs all assistant physicians and attendants, and prescribes their powers and duties. (Idem, §§ 835–841.)

Inquiry as to Lunacy.—Five or more persons, not more than one of whom is a relative of an alleged insane person, may petition the county judge or a judge of the Circuit Court having jurisdiction, for the examination of a person deemed insane. The judge to whom the petition is presented shall appoint two intelligent citizens, neither of whom is a petitioner, and one practising physician, as an examination committee. Such committee shall secure the presence of the supposed insane person, make a thorough examination, and determine as to the insanity of such person. If such per-

son is declared insane, the committee shall state whether the insanity is chronic or acute, its apparent cause, the hallucination if any, and the age and propensities of the subject; also whether indigent or possessing sufficient available estate for his support. A hearing may be had on petition of the alleged insane person. The report of the committee is to be signed by the members thereof, and presented to the judge before whom the proceedings were instituted. If the judge is satisfied from the report that the person is insane, he shall make an order directing the sheriff to deliver the insane person to the superintendent of the Florida Asylum. If the insane person is harmless and incurably insane, he may be ordered to be delivered to the county commissioners for care and maintenance. (Laws of 1895, ch. 4357, p. 123.)

Appointment and Powers of Guardians.—Guardians of insane persons are appointed by judges of the Circuit Court. No such guardian shall be appointed until the ward's insanity has been judicially declared. The powers and duties of such guardians are the same as those of the guardians of minors. (Idem, ch. 4359, p. 126.)

Guardians may make contracts relative to the person and estate of lunatics, but before such contracts shall bind the estate they must be confirmed by the Circuit Court. The real estate may be sold on application of their guardians in the same manner as the real estate of infants. (Revised Statutes of Florida, §§ 2110, 2111.)

GEORGIA.

(State Sanitarium, Milledgeville.)

The terms "lunatic," "insane," and *non compos mentis* each include all persons of unsound mind. (Code of Georgia, § 5.)

Courts of Ordinary have authority to exercise original, exclusive, and general jurisdiction of the following subjects:

5. The appointment and removal of guardians of persons of unsound mind.

6. All controversies as to right of guardianship. (Idem, § 4232.)

Guardians.—The ordinaries of the several counties of this State may appoint guardians for idiots, lunatics, and insane persons, deaf and dumb persons, when incapable of managing their estates, habitual drunkards, or persons imbecile from old age or other causes and incapable of managing their estates. (Idem, § 2570.)

Guardians shall take the same oath and give a like bond as guardians of minors, and their powers, duties, and liabilities shall be the same and be exercised under the same rules and regulations. (Idem, § 2571.)

A wife shall in all cases be entitled to preference in the appointment as guardian. (Idem, § 2572.)

Commission to Inquire as to Lunacy.—A commission may be granted upon the petition of any person. The commission shall be directed to eighteen proper persons, one of whom shall be a physician. It requires any twelve of them, including the physician, to examine the person by inspection for whom guardianship or commitment to the asylum is sought, to hear and examine witnesses as to his condition and capacity to manage his estate, and to make return of such examination to the said ordinary, specifying in such return into which of said classes they find the person to come. Upon such return, if the person be found insane, the ordinary shall appoint a guardian or commit him to an asylum. An appeal may be had by persons

dissatisfied and interested in the application for the commission upon bond, etc., to the Superior Court of the county. (Idem, §§ 2573–2575.)

Revocation of Guardianship.—Upon restoration to sanity and capacity of any person for whom a guardian has been appointed, such person may petition the ordinary praying the revocation of the guardianship. The ordinary shall examine into the truth, and if satisfied, and the guardian consenting thereto, the ordinary shall grant the prayer and deliver to such person his property and effects. If the ordinary is not satisfied as to the truth of the petition, the question shall be tried before a jury. (Idem, §§ 2578, 2579.)

Confinement of Ward.—Guardians of insane persons are authorized to confine them if such a course is necessary, either for their protection or for the safety of others; and a guardian wilfully failing to take such precaution with his ward shall be responsible for injuries inflicted on others by such ward. Where there is no guardian, or the guardian refuses to confine his ward, and any person shall make oath that such insane person should not longer be left at large, the ordinary shall issue a warrant for the arrest of such person, and on an investigation of the facts may commit him to the lunatic asylum, and if necessary cause him to be temporarily confined in a jail. (Idem, §§ 2581, 2582.)

Expenses of Proceedings; How Paid.—Where the estate of an insane person is insufficient to defray the expenses of conducting the proceedings inquiring as to his lunacy and for carrying or conveying such insane person from the county to the State Lunatic Asylum, when committed to such asylum, then the county shall defray such expenses. (Idem, § 2583.)

Guardians of Persons in Lunatic Asylum.—Guardians may be appointed for inmates of the lunatic asylum, without trial, when it appears that such inmates are confined there in pursuant to a commitment. (Idem, § 2584.)

Georgia State Sanitarium.—This is managed by ten trustees appointed by the governor. The trustees appoint all the officers annually, and may remove them for misconduct. They prescribe rules for the admission and discharge of lunatics, epileptics, idiots, and demented inebriates who are received and maintained at State expense. Harmless and incurable patients may be remanded to the county authorities having charge of county paupers. (Laws of 1897, p. 109.)

All physicians appointed for the sanitarium are subjected to examination by the board of trustees, are appointed for two years, and are removable at the pleasure of the trustees.

Admission and Discharge of Patients.—Lunatics, idiots, epileptics, and demented inebriates may become inmates of the sanitarium. The patients are classified as: 1. Pay or pauper patients residents of this State; 2. Pay patients being non-residents; 3. Insane penitentiary convicts; 4. Insane negroes in certain cases. Negroes and whites are separated, as also are males and females and penitentiary convicts. If at the time of application for commitment, all cannot be accommodated, residents of Georgia are to be preferred. Pay patients may be admitted if accompanied by authentic evidence of insanity, or there is produced a certificate of three physicians, acquainted with the condition of the patient, or from one physician and two citizens, stating the cause of the application. (Code of Georgia, §§ 1429–1436.)

Pauper patients are supported at the expense of the State. If the patient is possessed of property sufficient for his support, such support shall be taken

from such property. If others are bound by law to support the patient, the State may recover from them the amount expended. (Idem, §§ 1439, 1440.)

Trial of Lunacy.—A friend or relation of a patient in the sanitarium, or the patient himself, may demand of the superintendent a trial by jury of the question of lunancy. The person demanding the trial must make an affidavit that the alleged cause of commitment did not or does not exist, and that the conviction was obtained by fraud, collusion, or mistake. (Idem, §§ 1443, 1444.)

Insane Negroes.—Separate apartments are provided for insane negroes. They are supported in the same manner as other patients. (Idem, § 1445.)

Insane Convicts.—Insane convicts are received on the certificate of the penitentiary physician and the principal keeper, stating the insanity of the convict. (Idem, § 1446.)

IDAHO.

(Idaho Insane Asylum, Blackfoot.)

When it appears to the satisfaction of a magistrate of the county that any person within the county is so far disordered in mind as to endanger health, person, or property, he shall issue and deliver to some peace officer for service a warrant directing that such person be arrested and taken before any judge of a court of record within the county for examination. The judge shall issue subpœnas for at least one graduate of medicine to attend such examination. The physician shall hear testimony and make a personal examination of the alleged insane person. If the physician believes the person to be dangerously insane, he shall make a certificate in his own handwriting showing, as near as possible: 1. That such person is so far disordered in his mind as to endanger health, person, or property. 2. The premonitory symptoms, apparent cause, or class of insanity, and the condition of the disease. 3. The nativity, age residence, occupation, and previous habits of the person. 4. The place whence the person came and the length of his residence within the State. The judge, after such examination and certificate, if he believes the person so disordered, shall make an order confining him in the insane asylum. (Statutes of Idaho, 1887, §§ 769–776.)

The insane person, together with the certificate of the physician and the order of the judge, is delivered to the sheriff; the sheriff shall immediately notify the superintendent of the asylum, who shall designate some person among the asylum employés to transport such insane person to the asylum. The expense of transporting patients is a charge against the asylum. (Idem, §§ 777, 780, as amended by Laws of 1895, p. 17, and Laws of 1899, p. 114.)

The physician attending such examination is allowed five dollars, to be paid by the county treasurer on the order of the Board of County Commissioners. (Idem, § 781.)

The Idaho Insane Asylum is located at Blackfoot, and is under the management and control of a board of directors consisting of three persons. The medical superintendent shall be a graduate of medicine, and have practised his profession within five years after the date of his diploma. He shall reside at the asylum and give his entire time and attention to promoting the best interests of the patients. He is the chief executive officer of the asylum, and shall control the patients, prescribe the treatment, and prescribe and enforce the sanitary regulations of the asylum. (Idem, §§ 750, 758.)

Any person received in the asylum shall after recovery be discharged therefrom.

No person suffering from contagious or infectious diseases shall be admitted into the asylum as a patient. (Idem, §§ 764, 767.)

ILLINOIS.

(Illinois Central Hospital for the Insane, Jacksonville; Illinois Northern Hospital for the Insane, Elgin; Illinois Southern Hospital for the Insane, Anna; Illinois Hospital for Insane Criminals, Menard; Illinois Asylum for the Incurable Insane, Bartonville; Cook County Hospital for the Insane, Dunning; Illinois Eastern Hospital for the Insane, Hospital; Illinois Western Hospital for the Insane, Watertown.)

The word insane in this Act shall be construed to mean any person who, by reason of unsoundness of mind, is incapable of managing and caring for his own estate, or is dangerous to himself or others if permitted to go at large, or is in such condition of mind or body as to be a fit subject for care and treatment in a hospital or asylum for the insane; *provided* that no person, idiot from birth, or whose mental development was arrested by disease or physical injury occurring prior to the age of puberty, and no person who is afflicted with simple epilepsy, shall be regarded as insane, unless the manifestations of abnormal excitability, violence, or homicidal or suicidal impulses, are such as to render his confinement in a hospital or asylum for the insane a proper precaution to prevent him from injuring himself or others. (Myers, Statutes of Illinois, 1897, chap. 85, § 1.)

Inquisition as to Lunacy.—When any person is supposed to be insane, any respectable citizen residing in the county may file a statement with the clerk of the County Court for proceedings to inquire into such alleged insanity. Upon the filing of such statement, the judge shall order the clerk of the court to issue a writ directed to the sheriff or any constable, or person having the custody or charge of such person, requiring the alleged insane person to be brought before him at a time and place to be appointed. Such writ need not be issued if the person alleged to be insane appears in court, or if it appear by affidavit of some credible person that it would be harmful to such person to be brought into court. The hearing shall not take place until the person alleged to be insane has received notice thereof. (Idem, §§ 3, 4.)

Trial shall be by jury of six persons one of whom shall be a physician. When no jury is demanded or it appears to the judge that a jury trial would be harmful to the alleged insane person, the judge shall appoint a commission of two physicians, chosen for their known ability and integrity, to examine and report the condition of the alleged insane person. (Idem, §§ 5-7.)

Inquests may be in open court, in chambers, or at the home of the person alleged to be insane, at the discretion of the court; the judge shall preside, and the presence of the patient shall be indispensable and no proceedings shall be had in his absence. The jury or commission shall furnish to the court written answers to such interrogatories as may be contained in a form prescribed by the State Commission of Public Charities. The court may set aside the findings of the jury or commission. The finding shall be recorded in full, and the judge shall enter his order in accordance therewith. Such order may discharge the patient, remand him to the custody of his friends, commit him to a public or private hospital or asylum, or to a county insane asylum, or insane department of a county almshouse. (Idem, §§ 8-11.)

Maintenance in State Hospital.—All insane persons admitted to a State hospital are maintained and treated at the expense of the State. The cost of clothing, transportation, and other incidental expenses is a county charge, if not paid by the patient or his relatives. (Idem, § 15.)

Admission of Patients to Hospital.—Upon entry of an order of commitment of an insane person to a State hospital the clerk of the court shall furnish a copy of the findings of the jury or commission, and interrogatories and answers, to the superintendent, who shall at once admit the patient. If there is no vacancy in such hospital, the statute provides for a return to the county of a harmless chronic insane person. (Idem, § 17.)

Discharge of Patient.—The board of trustees of each hospital has the power to discharge a patient, but such power may be delegated to the superintendent. Such discharge may be made because the person adjudged insane is not insane, or has recovered, or is improved so as to be able to care for himself, or his friends request his discharge, and in the opinion of the superintendent no evil consequences are likely to follow. Patients may also be released on parole for a term not exceeding three months. Discharged patients are entitled to a sum of money sufficient to pay their expenses to their homes and to provide them with proper clothing. (Idem, §§ 22, 23.)

State Board of Public Charities.—The State Board of Public Charities is charged with enforcement of the laws relating to the insane. (Idem, § 33.)

Appointment of Conservator.—When a person is adjudged insane, and he is possessed of any property which, in the opinion of the court, is in danger of waste or depreciation, he may, on the application of the person instituting the proceedings, appoint some fit person to be the conservator of such insane person; but in case there is a Probate Court in the county, then he shall transmit thereto a duly certified copy of the record of the verdict of the jury, or report of the commission, finding such person insane, and upon presentation the Probate Court may appoint some fit person to be conservator of such insane person. (Idem, § 12.)

Whenever any idiot, lunatic, or distracted person has any estate, real or personal, or when any person by excessive drinking, gaming, idleness, or debauchery of any kind, so spends, wastes, or lessens his estate as to expose himself or his family to want or suffering, the County Court of the county in which such person lives shall, on the application of any reputable citizen living in such county, proceed to ascertain whether such person be an idiot, lunatic, a drunkard, or such spendthrift, in the manner prescribed for conducting an inquest of lunacy. If it be so ascertained, the court shall appoint a conservator for such person. If there be a Probate Court in such county, the conservator must be appointed by the judge thereof on the proceedings being reported to him. (Idem, ch. 86, §§ 1, 2.)

Powers and Duties of Conservator.—The conservator shall give a bond in double the amount of his ward's real and personal estate, which may be put in suit in the name of the people of the State of Illinois to the use of any person entitled to recover on a breach thereof. He shall have the care and management of the estate of his ward and the custody of his person until otherwise ordered by the court, and the custody and education of his children where no other guardian is appointed, but shall not deprive the mother of the custody and education of the children without her consent. He shall take charge of the estate of the ward, and within sixty days return to the court a true and perfect inventory of the estate of the ward, signed and verified by him. He shall at the expiration of a year settle his ac-

counts as conservator with the County Court. He shall at the expiration of his trust pay and deliver to those entitled thereto all property in his hands as conservator in such manner as the court may direct. On every accounting or final settlement he shall set forth specifically on what account expenditures were made by him, and all sums received and paid out since his last accounting, and show the true balance of money on hand, which account shall be accompanied by proper vouchers and signed and verified by him. He shall deliver all accounts of his ward, and demand and sue for and receive in his own name as conservator all property of and demands due the ward, and may compromise the same with the approbation of the court. (Idem, §§ 3-11.)

He shall manage the estate of his ward frugally and without waste, and apply the income to the support of his ward and family and the education of his children. He shall invest the property of his ward in securities approved by the court. He may lease the ward's estate on terms and for a length of time approved by the court. He may, by leave of the court, mortgage the real estate of the ward upon petition to the County Court setting forth the condition of the estate and the facts and circumstances, which shall be published in a newspaper in the county for at least once a week for three successive weeks. The County Court may order the real estate of the ward to be sold in the manner and at a time and place prescribed by the court. The conservator making such sale shall make a return thereof to the court; and if the sale be approved by the court, the title shall vest in the purchaser of the estate so sold. (Idem, §§ 17-29.)

Removal of Conservator.—A conservator may be removed by the County Court for failure to give bond, for failure to make inventory, or to account and make a settlement, or if he shall have become insane, or have moved from the State, or become incapable or unsuitable for the discharge of his duties, or for failure to discharge any duty required of him by law or order of the court. Before removing a conservator the court shall summon him to show cause. When it appears proper, the court may permit the conservator to resign if he shall first settle his accounts and deliver over the estate as the court directs. (Idem, §§ 32-34.)

Appointment of Successor.—Upon the removal, resignation, or death of conservator, another shall be appointed. The court may compel the conservator so removed or resigned, or the executor or administrator of a deceased conservator, to deliver up to such successor all the property and effects in his custody or control belonging to the ward. (Idem, § 35.)

Compensation.—A conservator on settlement shall be allowed such fees and compensation for his services as shall seem reasonable and just to the court.

Restoration to Reason.—When any person for whom a conservator shall have been appointed shall be restored to his reason, or, if a drunkard or spendthrift, shall have reformed, such person may apply to the County Court to have said conservator removed and the care and management of his property restored to him. Notice of such application shall be given to the conservator ten days before the commencement of the term of the court to which application shall be made. A jury shall be summoned to try the question whether said applicant is a fit person to have the care, custody, and control of his property. If they return that such person is a fit person, then the court shall enter an order fully restoring such person to all the rights and privileges enjoyed before such conservator was appointed. (Idem, § 37-39.)

Appeals.—Appeals shall be allowed to the Circuit Court, from any order or judgment under this Act, upon the appellant giving bonds directed by the court.

INDIANA.

(Central Indiana Hospital for the Insane, Indianapolis; Eastern Indiana Hospital for the Insane, Richmond; Northern Indiana Hospital for the Insane, Logansport; Southern Indiana Hospital for the Insane, Evansville.)

The words "person of unsound mind" as used in this Act or any other statute of this State, shall be taken to mean any idiot, *non compos*, lunatic, monomaniac, or distracted person. (Revised Statutes of Indiana, 1852, p. 333, § 1; Burns, Statutes, § 2714.)

Inquisition as to Unsound Mind.—Whenever any person shall by statement in writing represent to the court having probate jurisdiction in any county, that any inhabitant of such county is a person of unsound mind and incapable of managing his estate, such court shall cause such person to be produced in court, and shall cause an issue to be made by the clerk of such court, denying the facts set forth in such statement; which issue shall be tried by a jury to be empanelled under the direction of such court. It is the duty of the prosecuting attorney to appear for the person alleged to be insane. If the jury finds such person to be of unsound mind, the court shall appoint a guardian for such person, who shall have the custody of his person and the management of his estate, and shall give a bond and take an oath in the manner required of guardians of minors. If such insane person is a pauper, he shall be provided for under the laws regulating the relief of the poor. If being a pauper he shall be dangerous, he shall be provided for under the regulations prescribed in section 12. If the court shall be satisfied that such person alleged to be of unsound mind cannot without injury to his health be produced in court, such personal appearance may be dispensed with. (Idem, §§ 2, 3, 4, as amended by Laws of 1895, ch. 99; Burns, Statutes, §§ 2715–2717.)

Duties and Powers of Guardians.—The same duties are required of, and the same powers granted to, guardians of persons of unsound mind as are required of and granted to guardians of minors so far as the same may be applicable. (Idem, § 8; Burns, Statutes, § 2721.)

Termination of Guardianship.—Guardianship shall terminate upon the restoration to reason or the death of the ward. Whenever it shall be alleged that an insane person has become of sound mind, the fact may be tried and determined in the same manner as the allegation of the unsoundness of mind, and the expenses of such trial shall be paid by such guardian out of the estate of the ward. (Idem, §§ 9, 10; Burns, Statutes, §§ 2722, 2723.)

Dangerous Insane.—If it shall appear to the court that there is danger to the community in permitting an insane person to run at large, the court shall make such order for his safekeeping as may be necessary, and direct the expenses thereof to be paid out of the estate of such person; or if that be insufficient or necessary for the support of his family, then out of the county treasury. (Burns, Statutes, § 2525.)

Arrest and Commitment of Dangerous Insane Persons.—A justice of the peace shall issue a warrant to any constable or to the sheriff, commanding him to apprehend and bring before him any person alleged upon oath to be insane and dangerous to the community if suffered to remain at large; he may subpœna such witnesses as may be demanded by either party

under the same regulations as govern criminal prosecutions in justices' courts. Immediately after the issue of such warrant he shall cause a jury to be empanelled and summoned. Such justice shall interrogate each of such jurors under oath as to whether he is prejudiced against such insane person, has any interest in his property, or is related to him or any member of his family within the fifth degree. If he answer affirmatively, he shall be rejected. Such insane person or any person in his behalf shall have the right to challenge three of such jurors peremptorily. If the jury find after hearing the evidence that such person is not insane, he shall be discharged from custody, and the costs of such proceeding shall be taxed against the complaining party; but if the jury find that such person is insane and dangerous to the community if suffered to remain at large, such justice shall appoint some resident of the county to take charge of and confine such person, for which he shall receive a reasonable compensation, to be paid out of the county treasury upon the order of the board of commissioners. Such commissioners may appoint some other person to take charge of such insane person at any time in their discretion. Such justice, in case the finding of the jury be against such insane person, shall certify his proceedings within ten days thereafter to the Circuit Court of the county. At the next term of such court, after the filing of the transcript of the proceedings in the clerk's office thereof, such court shall cause the issue to be again tried by a jury of twelve persons, under like instruction and conditions as to the manner of empanelling the jury, etc., as hereinbefore provided; but if the finding of such jury shall be against such insane person, the court shall confirm the appointment of the person appointed to take charge of the insane person or may appoint some other suitable person for that purpose. (Laws of Indiana, 1855, p. 133, §§ 1–5; Burns, Statutes, §§ 6987–6991.)

Application of Estate of Insane Person.—If such insane person have a family and his property does not exceed in value five hundred dollars, no part of his estate shall be applied to the payment of the costs of such proceeding or for his keeping; but when he shall have no family all his property, or where he has a family and his property shall exceed in value five hundred dollars, the excess thereof above five hundred dollars shall be subject to the payment of said costs and expenses of keeping, and the court shall order whatever sum has been paid out upon the order of the board of commissioners to be refunded out of his estate to the county treasury, and shall make provision out of such estate for the payment of expenses to afterward accrue, until such estate is exhausted or such insane person is discharged. (Idem, § 6; Burns, Statutes, § 6992.)

Writ of Habeas Corpus.—Any person committed as insane to any State hospital may apply to the proper authorities for a writ of habeas corpus, and the question of insanity shall be decided at the hearing, and an adverse decision shall not operate as a bar to the issuance of another writ; provided, however, that writs of habeas corpus shall not issue oftener than once in every period of three months.

As to the management of the State Hospitals for the Insane and the admission of patients thereto, see Acts of Indiana, 1881, §§ 2835–2879; Burns, Statutes, 1894, §§ 3201–3250.)

Insane Convicts.—The wardens of the State prisons are required to report cases of insanity to the governor. The governor thereupon orders the warden to convene a lunacy commission to determine the question of insanity, who, upon completing their examination, report to the warden, and the warden transmits the report to the governor. The governor, if the con-

vict is declared insane, directs his removal to a State hospital. If the patient is cured, the superintendent of the hospital is required to report the fact to the governor, who notifies the warden, and the convict is returned to prison if his term has not expired. (Statutes of Indiana, Laws of 1895, ch. 83.)

IOWA.

(Hospital for the Insane, Mt. Pleasant; Hospital for the Insane, Independence; Hospital for the Insane, Clarinda; Hospital for the Insane, Cherokee; Mercy Hospital for the Insane, Davenport.)

The words "insane person" include idiots, lunatics, distracted persons, and persons of unsound mind. (Iowa Code, 1897, § 48, subd. 6.)

The term "insane," as used in this chapter, includes every species of insanity or mental derangement. The term "idiot" is restricted to persons foolish from birth, supposed to be naturally without mind. No idiot shall be admitted to a State hospital. (Idem, 1897, § 2298.)

Guardians of Drunkards, Spendthrifts, and Lunatics.—Appointment of guardians is vested in the Circuit Court. When a petition is presented to the Circuit Court, verified by affidavit, that any inhabitant of the county is: 1. An idiot, lunatic, or person of unsound mind; 2. An habitual drunkard, incapable of managing his affairs; 3. A spendthrift who is squandering his property; and the allegations of the petition are satisfactorily proved upon trial, the court may appoint a guardian of the property, who shall also be a guardian of the minor children. The petition shall set forth the facts upon which the application is based, to be answered as in other ordinary actions. The applicant shall be recorded as plaintiff, the other party as defendant, and either party may demand a trial by jury. (Idem, §§ 3219, 3220.)

Custody.—Priority of claim to the custody of any such person shall be: 1. The legally appointed guardian. 2. The husband or wife. 3. The parents. 4. The children. (Idem, § 3228.)

Powers and Duties of Guardians.—The provisions of this Code relating to guardians for minors and regulating or prescribing the powers, duties, or liabilities of each, and of the court so far as the same are applicable, shall be held to apply to guardians and their wards appointed under the foregoing sections. A guardian may sue in his own name as guardian of the ward for whom he sues. Action shall not abate upon the termination of his guardianship; but his successor or the person for whom he was guardian shall be made a party to the suit in the same manner as an executor or administrator is made a party to a proceedings of the like kind. A guardian may sell real estate of the ward for the support of the ward or his family and for the payment of his debts under like proceedings as required by law for the sale of real estate of minors. If the estate of any such person is insolvent, a like proceeding may be had as is required by law for the settlement of the insolvent estate of a deceased person. (Idem, §§ 3223–3227.)

Confinement of Lunatics.—Hospitals are established for the care of the insane at Mt. Pleasant, in Henry County, at Independence, in Buchanan County, and at Clarinda, in Page County, and at Cherokee, each under the charge and management of five trustees, two of whom may be women, and one of whom shall be a resident of the place where the hospital is located. (Idem, 1897, § 2253.)

Commissioners of Insanity.—In each county there is a board of three commissioners of insanity, composed of the clerk of the District Court, and

two others appointed by the judge of the court, one of whom shall be a physician and the other a lawyer. Such commissioners have cognizance of all applications for admission to a hospital or for the safekeeping otherwise of insane persons within their counties. They may issue subpœnas, compel obedience thereto, administer oaths, and do any act of a court necessary and proper in the premises. (Idem, §§ 2261-2263.)

Commitment to Hospital by Commissioners.—Application for admission to a hospital must be made by information verified by affidavit alleging that the person in whose behalf the application is made is a fit subject for custody and treatment in the hospital; that such person is a resident of the county or has a local settlement therein, if such is known to be the fact, and if such settlement is not in the county where it is, if known. The commissioners shall thereupon examine the informant under oath, and if satisfied that there are reasonable grounds shall at once investigate. They may require the person for whom such admission is sought to be brought before them. They may provide for the suitable custody of such person until the determination of their investigation. They shall hear testimony for and against such application. Any citizen of the county or relative of the person alleged to be insane may appear and resist the application. The commissioners shall appoint some regular practising physician of the county to make a personal examination of the person alleged to be insane, touching the truth of the information, and report forthwith to them. Such physician so appointed shall certify that he has made a careful personal examination; that he finds the person in question insane, if such is the fact. On the return of the physician's certificate the commissioners shall conclude their investigation, and shall find whether the person be insane; and if insane, whether he is a fit subject for treatment and custody in the hospital. If they find such person is not insane, they shall order his immediate discharge. If found insane, they shall order such person to be committed to the hospital, and shall forthwith issue their warrant and a duplicate thereof, stating such finding and authorizing the superintendent of the hospital to receive and keep such person as a patient therein. Such warrant and duplicate, with the certificate and finding of the physician, shall be delivered to the sheriff of the county, who shall execute the same by conveying such person to the hospital. No female shall be taken to a hospital without the attendance of some other female or some relative. The warrant may be executed by a relative or immediate friend of the patient. The superintendent of the hospital shall acknowledge such delivery on the original warrant, which the sheriff shall return to the clerk of the commissioners. No person during such investigation or while being conveyed to the hospital shall be confined in any jail or prison except in cases of extreme violence, when it is deemed absolutely necessary for the safety of such insane person and of the public that he be confined in such jail or prison; but at no time shall any female be placed in such confinement without at least one female attendant remaining in charge of such insane person. (Idem, 1897, §§ 2264-2266.)

Appeal from Finding.—Any person found insane by the commissioners of insanity may appeal to the Circuit Court within ten days after the filing of the finding of such commissioners; the case when thus appealed shall be placed upon the docket by the clerk of the court and stand for trial anew in such court. If any person found insane takes an appeal from such finding, such person shall be discharged from custody pending such appeal, unless the commissioners for any reason find that such person cannot with safety be

allowed to go at large. If upon the trial of such an appeal such person is found not insane, the court shall order the immediate discharge of such person if in custody. If found insane, the court shall order such person to be committed to the hospital, and the clerk of the court shall issue a warrant to carry such finding and order into effect. (Idem, §§ 2267–2269.)

Custody Outside of Hospital.—If any person found to be insane cannot be at once admitted to a hospital for want of room, or for any other cause, and cannot with safety be allowed to go at liberty, the commissioners shall provide otherwise for the custody of such patient outside of the hospital until the occasion therefor no longer exists. Such patients may be cared for as private patients, if relatives or friends are willing to provide for them without public charge. In such cases the commissioners may appoint special custodians with authority to restrain, protect, and care for such patients. (Idem, 1897, § 2271.)

Discharge from Hospital.—Upon the application of the relatives or friends of any patient in the hospital who is not cured, and who cannot be safely allowed to go at liberty, the commissioners may provide for the care of such patient within the county. Whenever it shall be shown to the satisfaction of the commissioners that cause no longer exists for the care within the county of any particular person as an insane patient, they shall order the immediate discharge of such person. (Idem, §§ 2276, 2277.)

Expenses of Maintenance at Hospital.—The county of settlement of the insane person is liable to the cost and expense of the maintenance of such patient within the hospital. Patients in the hospital having no local settlement within the State, or whose local settlement cannot be ascertained, shall be supported at the expense of the State. All patients in the hospital shall be regarded as standing upon an equal footing, and the several patients, according to their different conditions of mind and body, and their respective needs, shall be provided for and treated with equal care; but if the relatives or friends of any patient shall desire and shall pay the expenses therefor, such patient may have special care, and may be provided with a special attendant, as may be agreed upon with the superintendent. In such case the charges for such special care and attendance shall be paid quarterly in advance. (Idem, §§ 2281, 2283–2285.)

Discharge when Cured.—Any patient who is cured shall be immediately discharged by the superintendent. Upon such discharge the superintendent shall furnish the patient, unless otherwise supplied, with suitable clothing, and a sum of money not exceeding twenty dollars, which shall be charged with the other expenses in the hospital to such patient. (Idem, § 2288.)

Visiting Committee.—The governor shall appoint a visiting committee of three to visit the insane asylums of the State at their discretion, and without giving notice of their intended visit. They may go through the wards unaccompanied by any officer of the institution, may send for persons and papers, and examine witnesses on oath to ascertain whether any of the inmates are improperly detained in the hospital or unjustly placed there, and whether they are kindly treated, with full power to correct any abuses found to exist. They may discharge any attendant or employé who has been guilty of inflicting injury upon the inmates of the hospital. (Idem, § 2291.)

Privileges of Inmates.—The names of the visiting committee and their post-office address shall be kept posted in every ward in the asylum, and every inmate in the asylum shall be allowed to write once a week to

this committee; and any member of the committee neglecting to heed the call of a patient to him for protection, when proved to have been needed, shall be deemed unfit for his office, and shall be removed by the governor. Every person confined in an insane asylum shall be furnished by the superintendent, or party having charge of such person, at least once in each week, with suitable materials for writing and mailing letters, if they request the same, unless otherwise ordered by the visiting committee, which order shall continue in force until countermanded by said committee. The superintendent shall receive, if requested to do so by the person so confined, at least one letter in each week, addressed to one of the visiting committee, without opening or reading the same, and shall without delay deposit it in the post-office for transmissal, with the proper postage-stamp affixed thereto, but all other letters may be examined by the superintendent, and if in his opinion it seems proper he may retain the same. (Idem, §§ 2300–2302.)

Writ of Habeas Corpus.—All persons confined as insane shall be entitled to the benefit of the writ of habeas corpus, and the question of insanity shall be decided at the hearing, and if the judge shall decide that the person is insane, such decision shall be no bar to the issuing of the writ a second time. (Idem, § 2306.)

KANSAS.

(Osawatomie State Hospital, Osawatomie; Topeka State Hospital, Topeka.)

Care and custody of the person and property of idiots, lunatics, or habitual drunkards, are vested in the Probate Courts.

Inquisition as to Lunacy.—Upon information to the Probate Court that any person is an idiot, lunatic, or person of unsound mind, or habitual drunkard, and incapable of managing his affairs, if satisfied that there is good cause, the court shall cause the facts to be inquired into by a jury. The court may cause the person to be brought before the court. Any justice of the peace, sheriff, coroner, or constable, discovering any person to be of unsound mind or a habitual drunkard, shall make application to the Probate Court for the exercise of its jurisdiction. (General Statutes of Kansas, ch. 60, §§ 1–4.)

Trial of Inquisition.—A jury of six persons, one of whom shall be a physician in regular practice and good standing, shall be empanelled to try the case. The person alleged insane may be present at the trial, assisted by counsel, and may challenge jurors. After hearing the evidence the jury shall render their verdict in writing, signed by them, embodying the substantial facts shown by the evidence in form prescribed by statute. There shall be attached to the verdict a brief statement of the medical treatment in the case, together with any other information or circumstances known which will throw light on the case, to be signed by the physician upon the jury. The verdict shall be recorded at large by the probate judge; and if it appear that the person is insane and fit to be sent to an insane asylum, the court shall order him committed to the State Insane Asylum, and application shall thereupon be made by the judge to the superintendent for the admission of such person, and the judge shall transmit with the application a copy of the verdict of the jury and the statement of the physician. If it be found that the person is incapable of managing his affairs, a guardian shall be appointed by the court of the person and estate of such person. (Idem, § 5.)

Costs.—If the person is found insane or an habitual drunkard, the costs

are payable out of his estate, or if that be insufficient by the county. If the person be discharged, the costs shall be paid by the person instituting the proceedings. (Idem, §§ 6, 7.)

Guardian.—The guardian of a person of unsound mind or an habitual drunkard shall give a bond approved by the Probate Court conditioned for the proper care of such insane person or habitual drunkard and the proper management of his estate and effects, and the faithful performance of all matters pertaining to the guardianship. The court may, at any time, require the guardian to give a new bond or additional security for any purpose. The bond shall be deposited with the court making the appointment. Every guardian shall within thirty days after his appointment publish a notice thereof. He shall take charge of the person committed to his charge and provide for his support and maintenance. He shall collect and take into his possession all the property of the person. Within three months after his appointment he shall make out and file in the office of the Probate Court a true inventory of the estate of his ward, containing a statement of the income and proceeds thereof, and the debts, credits, and effects. Upon the discovery of any new property, he shall file a like inventory. All such inventories shall be verified. He shall prosecute and defend all actions in which the ward is interested, shall collect all debts, and adjust and settle all demands due or becoming due from his ward. (Idem, §§ 9–18.)

Sale of Property of Ward. The property of the ward may be sold upon the order of the court for the support and maintenance of the ward's family and the education of his children. If the personal property of the insane person or habitual drunkard is insufficient for the payment of his debts, the support and maintenance of his family and himself, and the education of his children, the guardian shall petition the Probate Court praying authority to mortgage, lease, or sell as much of the real estate as shall be necessary to supply the deficiency. If it appear to the court that the personal estate is insufficient, the court shall make an order directing such mortgage, lease, or sale. Such order shall contain the time and terms of sale; or if a mortgage or lease be ordered, the terms of such mortgage or lease, and the manner in which the proceeds shall be secured and the income thereof appropriated. Such sale shall be upon notice published for four weeks. No real estate shall be sold at private sale for less than three-fourths of its appraised value. The guardian shall sell at the time and place appointed all such lands at public auction to the highest bidder, and shall make a verified report of the proceedings to the court, which report shall also state that the guardian did not directly or indirectly become the purchaser of the property sold, and that he was in nowise interested in the purchase thereof. If the court approve the proceedings, a deed shall be executed by the guardian to the purchaser. If the report be disapproved, the court may set aside the sale and order all moneys paid to be refunded. (Idem, §§ 19–30.)

Restoration of Person to Sanity.—If any person shall allege in writing, verified by oath, that any person declared insane or an habitual drunkard has been restored to his right mind or to temperate habits, the court by which the proceedings were had shall cause the facts to be inquired into either by a jury or without a jury, as may seem proper. If it shall be found that such person has been restored, the guardian shall be discharged, and he shall immediately settle his accounts and restore to such person all things remaining in his hands belonging to him. (Idem, §§ 37, 38.)

Termination of Guardianship.—In case of the death of any such insane person or habitual drunkard, the power of the guardian shall cease,

and the estate shall descend and be distributed in the same manner as if such person had been of sound mind and temperate habits, and the guardian shall immediately settle his account and deliver the estate and effects. A guardian may be removed at any time by the Probate Court. When removed, such guardian shall immediately settle his accounts and render to his successor the estate and effects of his ward. (Idem, §§ 39–41.)

Confinement of Lunatic by Guardian.—If any person shall be furiously mad or so far disordered in his mind as to endanger his own person or that of others, the guardian shall confine him in some suitable place until proceedings can be had for the proper restraint, support, and safekeeping of such person. If there be no person having such charge, any judge of a court of record or any two justices of the peace may cause such insane person to be apprehended, and may employ any person to confine him in some suitable place until the Probate Court shall make further orders. The expenses attending such confinement shall be paid by the guardian out of his estate or by the person bound to provide for and support such insane person, or the same shall be paid out of the county treasury. (Idem, §§ 43–46.)

Confinement of Insane Person in the State Insane Asylum.—Any probate judge in this State may commit an insane person to the State Insane Asylum. If the person adjudged insane has sufficient means for his maintenance and that of his family (if he have one), the court shall order his guardian to pay for his maintenance out of the proceeds of the estate of such insane person. If the estate be insufficient, the court shall deliver to the board of county commissioners a certificate stating such facts, and thereupon an order shall be issued for the payment of the expenses of such maintenance out of the county treasury. Private patients may be placed in an asylum at private expense if the superintendent can conveniently receive them. In such cases the superintendent shall be presented with a certificate, signed by at least one practising physician, and also a certificate of the probate judge of the proper county, to the effect that such persons are insane (Idem, §§ 47–52.)

Removal and Discharge of Persons from Insane Asylum.—The person or court placing a patient in an asylum may remove him at any time, and the superintendent may discharge him in accordance with the by-laws of the asylum. No idiot or person laboring under any infectious or contagious disease shall be admitted into an asylum. When a patient is ordered discharged, the steward of the asylum shall immediately notify the probate judge. In case the patient is discharged not restored, he shall immediately issue his precept to the guardian of such person to remove him from the asylum at the expense of the county or person charged with his maintenance. If the patient is not removed within thirty days after discharge, the steward shall remove him at the expense of the county or person charged with his maintenance. If the person be discharged restored, the steward may send him home at the expense of the county or of the person charged with his maintenance. (Idem, §§ 54–56.)

KENTUCKY.

(Eastern Kentucky Asylum for the Insane, Lexington; Central Kentucky Asylum for the Insane, Lakeland; Western Kentucky Asylum for the Insane, Hopkinsville.)

Custody of Persons and Estates of Idiots and Lunatics.—Jurisdiction is vested in the Circuit and County Courts of the persons and estates

of idiots, lunatics, or those who from confirmed bodily infirmity are unable to make known to others by speech, sign, or otherwise, their thoughts or desires, and, by reason thereof, are incompetent to manage their estates, or those whose minds on account of any infirmity or stress of age have become so imbecile or unsound as to render them incompetent to manage their estates. (Statutes of Kentucky, § 2149.)

Sale of Real Estate.—Any Circuit Court, upon the application of a committee, may order the sale of such part of the real estate of an incompetent person as is necessary for the maintenance of such person and his family, and may settle and distribute the estate in the manner provided for the settlement and distribution of the estates of insolvent decedents. (Idem, § 2150.)

Appointment of Committee.—A committee shall be appointed after the inquest of a jury and the judgment of the Circuit or County Court declaring the person insane. (Idem, § 2151.)

Power of Committee.—Power and duties of a committee are in all respects the same as those of the guardian of an infant, except as to education. But the court may appoint a person other than the committee to take charge of the person, idiot, lunatic, or incompetent person when he is not confined in the asylum. (Idem, § 2153.)

Claims against the Estate.—Claims against the estate of a person incompetent to manage his estate shall not be allowed or paid until verified and proved in the manner prescribed for the proof of claims against the estate of deceased persons. (Idem, § 2155.)

Inquest; How Held.—An inquest to inquire into the question of insanity shall be held only in the Circuit Court, if one be in session at the time. If no Circuit Court be in session, the inquest may be held by any judge of a Circuit Court or by the presiding county judge. No inquest shall be held unless the person alleged to be of unsound mind is in court and personally in the presence of the jury. Such personal presence shall only be dispensed with upon the oath of two regular practising physicians that they have personally examined the individual charged to be of unsound mind, and that they verily believe him to be an idiot or lunatic or incompetent to manage his affairs, and that his condition is such that it would be unsafe to bring him into court. The oath prescribed by statute shall be administered to the jury, and the judge shall instruct the jury so as to enable them to decide the question whether the defendant is an idiot or lunatic. If on the return of the verdict the court is satisfied with the inquest, judgment shall be entered according to the finding. If a judge is of the opinion that the verdict is not sustained by the evidence, or is contrary to law, he shall set it aside and award a new inquest. All papers pertaining to the inquest shall be delivered to the clerk of the court and filed by him. Whenever it shall appear to the County or Circuit Court, from an affidavit filed, that the insane person has been restored, or that the inquest was false or fraudulent, the facts shall be inquired into by jury in open court, and all necessary orders and decrees made by the court. If the lunatic be sent to an asylum, the judge presiding at the inquest shall ascertain and draw up a brief history of the patient's case, embracing certain points, which shall be transmitted with a record of the inquest to the asylum. An inquest shall be held upon the application of the attorney for the Commonwealth or, if he be absent, of the county attorney. The court shall appoint some member of the bar to represent and protect the interests of the alleged insane person, and it shall be the duty of the attorney for the Commonwealth or county to

prevent the finding of any person as an idiot or lunatic, who in his opinion is not such; or the finding of any person an idiot who is a lunatic. (Idem, §§ 2156–2162.)

Confinement of the Insane.—The judge holding the inquest may by order commit a person found insane to a lunatic asylum, when it will be proper for the court to do so; but in no case shall an order be made sending such person to an insane asylum unless the jury by their verdict on the inquest shall find that he is so dangerous or uncontrollable that he cannot properly or safely be kept by a committee at home. (Idem, § 2156.)

Asylum for the Insane.—There shall be for each asylum a medical superintendent, and a first, second, and a third assistant physician, each of whom shall be skilful physicians, and a steward. The officers shall reside in the asylum. They are appointed by the governor, by and with the advice and consent of the senate. The medical superintendent shall have the general management, supervision, and control of the patients, subject to the regulations of the board of commissioners, and shall devote his entire time thereto. Mechanical restraint shall not be applied in any case without express direction and under the supervision of one of the physicians in charge of said hospital, nor shall restraining apparatus be kept in the wards when not in use. (Laws of 1894, ch. 48, art. 2, §§ 1–6.)

The Return of Idiots and Harmless Incurable Lunatics.—All pauper idiots, epileptics, and harmless incurable lunatics shall be returned by the asylum in which they are confined to the several counties whence they were sent. The president of the board of commissioners, or the superintendent and one other commissioner, shall act as a commission to pass upon such case as the superintendent may propose to send back. They shall investigate each case carefully, and if all concur that the inmate can be safely sent back, they shall order him returned to the county whence he was sent. The cost of returning pay patients shall be paid by their committee or relatives willing and able to pay, and the cost of returning pauper inmates and of pay patients from whose committee money cannot be collected therefor, shall be paid by the auditor upon the certificate of the superintendent. (Idem, §§ 238–244.)

There shall be at least one woman physician in each asylum in which female patients are maintained. Such woman physician shall possess the same rights as a male physician of like rank. (Laws of 1898, ch. 29.)

LOUISIANA.

(Louisiana Insane Asylum, Jackson.)

State Asylum.—The asylum for the insane is governed by a board of administrators, under the name and style of the "Board of Administrators of the Insane Asylum of the State of Louisiana." (Statutes of Louisiana, §§ 1760, 1761.)

Admission of Lunatic to Asylum.—Whenever it shall be made known to the judge of the District or Parish Court by petition or oath of any individual, that any lunatic or insane person within his district ought to be sent to or confined in an insane asylum in this State, such judge shall issue a warrant to bring such lunatic or insane person before him, and if after proper inquiry into all the facts and circumstances of the case he deems it necessary to confine such person in the asylum, he shall make out a warrant to a sheriff of the parish commanding him to convey the lunatic or insane person to the insane asylum. (Idem, § 1768.)

Examination by Physician of Asylum.—The physician of the asylum shall professionally examine the lunatic or insane person sent to the hospital by the authority of the district or parish judge, and if in his opinion said person is only feigning insanity, being a person charged with a felonious crime, he shall report to the board, which shall investigate the fact; and if, in the judgment of the majority, said person should not be admitted as an inmate of the asylum, the president of said board shall cause such person feigning insanity, and who had been previously committed to prison for a crime, to be confined in the parish jail, and shall immediately inform the president of the police jury of the parish, or the proper authority in the parish of Orleans where the rejected person has his domicile, of the fact and the reason of his rejection. The provisions of this section shall also apply to such persons charged with a crime who afterward recover and become sane in said asylum. (Idem, § 1776.)

Insane Convicts.—When a convict is insane, the warden of the penitentiary is required to apply to the District Court for his interdiction and removal from the penitentiary to the asylum for the insane. The judge before whom the application is made shall order the alleged insane convict to be brought before him for examination. If the convict is shown to be insane, he shall be removed to the asylum and detained until cured. (Laws of 1896, Act 105, p. 153.)

MAINE.

(Maine Insane Hospital, Augusta; Eastern Maine Insane Hospital, Bangor.)

The words "insane person" may include an idiot, *non compos*, lunatic, or distracted person.

Guardians of Insane and Incompetent Persons, Spendthrifts, and Convicts.—Any judge of probate may appoint guardians on written application of friends, relatives, or creditors, or of the municipal officers or overseers of the poor where they reside, for: 1. Persons insane or of unsound mind who, by reason of infirmity or mental incapacity, are incompetent to manage their estates or to protect their rights. 2. Persons who, by excessive drinking, gambling, idleness, or debauchery of any kind, have become incapable of managing their affairs, or who so spend or waste their estate as to expose themselves or families to want or suffering or their towns to expense. 3. Convicts committed to the State prison for a term less than for life. Guardians may be appointed for persons committed to the insane hospital without personal notice to the parties, and for insane or incompetent married women after personal notice and a hearing upon proof of the alleged insanity or incompetency, without inquisition by the municipal officers of the town. In all cases in which the municipal officers or overseers of the poor are applicants, if they have given at least fourteen days' notice to such person by serving him with a copy of their application, the judge may adjudicate thereon, without further inquisition, if such person is present, or on such further notice, if any, as he thinks reasonable; but if such officers have not given such notice, the judge shall cause personal notice to be given to the party before the hearing and adjudication. In all other cases the judge shall issue his warrant to the municipal officers of the town in which such person resides, requiring them to make an inquisition into the allegations made in the application. They shall decide whether such allegations are true, and as soon as may be report the result to the judge, and upon such report, after personal notice and a hearing, he may appoint a guardian. A copy of the application and the order of the court thereon shall be filed in the registry of

deeds for the county. When a guardian is thus appointed, the judge shall make an allowance from the ward's estate for all reasonable expenses of the guardian in defending himself against the complaint. Such guardians have the custody of the person of their ward, except so far as the Court of Probate may from time to time otherwise order; and every guardian appointed over any person for gambling, idleness, drinking, or debauchery, shall inculcate upon him habits of sobriety and industry, and when of sufficient health and strength, with the approbation of the judge, may bind him out to labor, not exceeding six months at any one time, or employ him in his own service. (Statutes of Maine, title 6, ch. 67, § 4.)

Powers and Duties of Guardians.—For the bond and the duties of a guardian as to the management of the ward's estate, and the powers of the guardian as to the purchase and sale of property and other matters pertaining to the care and management of the property of the ward, see Statutes of Maine, §§ 10–39, relating to the powers and duties, etc., of guardians of minors.

State Insane Hospital.—The government of the Maine Insane Hospital is vested in a committee of seven trustees, one of whom shall be a woman. Trustees may appoint a superintendent and a steward and treasurer subject to the approval of the governor and counsel, and all other officers necessary for the efficient and economic management of the business of the institution. There shall be, monthly by two of the trustees, quarterly by three, and annually by a majority of the full board, a thorough examination of the hospital. At each visit a written account of the state of the institution shall be drawn up by the visitors, recorded and presented at the annual meeting of the trustees, at which meeting they with the superintendent shall make a particular examination into the condition of each patient, and discharge any one so far restored that his comfort and safety and that of the public no longer require his confinement. (Idem, ch. 143, §§ 1–4.)

The government of the Eastern Maine Insane Hospital is vested in the trustees of the Maine Insane Hospital, who have the same powers in relation to such Eastern Hospital as is conferred upon them by Chapter 143 of the revised statutes in relation to the Maine Hospital. (Laws of 1899, ch. 75.)

Duties of Superintendent.—The superintendent shall be a physician, reside constantly at the hospital, have general superintendence of the hospital and grounds, receive all patients legally sent to the hospital, unless the number exceeds its accommodations, and have charge of them and the direction of all persons therein, subject to the regulation of the board of trustees. He shall apportion the number of patients who can be accommodated in the hospital among the towns according to their population by the last census; and when applications for admission exceed, or are liable to exceed, that number of patients, he shall give preference to those from towns that have not their full proportion of patients in the hospital, and may reject others. When a person has been unlawfully committed, the superintendent shall report the case to the trustees at their next monthly meeting, and they may cause the removal of such person to the town from which he was committed. The superintendent at each monthly visit of the trustees shall also report to them the name of any inmate who was idiotic at the date of his commitment, or who has become so imbecile as in his judgment to be beyond cure; and if he thinks that such inmate may be discharged with safety to himself and to the public, the trustees shall order his discharge and cause him to be removed to the town from which he was committed. (Statutes of Maine, title 6, ch. 143, §§ 7–9.)

Commitment of Insane Persons.—Parents and guardians of insane minors, if of sufficient ability to support them there, shall, within thirty days after an attack of insanity, without legal examination, send them to the hospital and give to the treasurer thereof the bond required. Insane persons not thus sent to any hospital shall be subject to examination as hereinafter provided. The municipal officers of towns shall constitute a board of examiners, and on complaint in writing of any relative or of any justice of the peace in their town they shall immediately inquire into the condition of any person in said town alleged to be insane. They shall take testimony, and if they think such person insane, or his comfort and safety and that of others will be promoted thereby, they shall send him to the hospital, with a certificate stating the fact of his insanity and the town in which he resided or was found at the time of the examination. The officers ordering the commitment of a person unable to pay for his support may, in writing, certify that fact to the trustees, and that he has no relatives liable and of sufficient ability to pay for it; and if the trustees are satisfied that such certificate is true, the treasurer of the hospital may charge to the State one dollar and a half a week for his board, and deduct it from the charge made to the patient or town for his support. (Idem, §§ 12–14; § 13, as amended by Laws of 1897, ch. 246.)

Appeal from Order of Commitment.—An appeal may be had by the person deeming himself aggrieved by the decision of the board of examiners within five days after the decision was rendered to the justices of the town. Such justices may thereupon take testimony and hear and decide the case. If they find the person insane, and believe that he will be more comfortable and safe to himself and others, they shall give a certificate for his commitment to the hospital like cases of no appeal. (Idem, §§ 15–18.)

Discharge of the Insane.—A friend, person, or town liable for the support of a patient who has been in the hospital for six months, not committed by the order of the Supreme Judicial Court, when afflicted with homicidal insanity, thinking that he is unreasonably detained, may apply to the municipal officers of the town where the insane person resides, and they shall inquire into the case and summon before them any proper witnesses, and their decision and order shall be binding on the parties. When the overseers of the town are notified by mail by the superintendent that the person supported at the hospital by the town has recovered, they shall cause him to be removed to their town. (Idem, title 6, ch. 143, §§ 23–25.)

MARYLAND.

(Mount Hope Retreat, Baltimore; Maryland Hospital for the Insane, Catonsville; Springfield State Hospital, Sykesville.)

Jurisdiction of Courts of Equity.—Courts of equity have power to superintend and direct the affairs of persons *non compos mentis* both as to the care of their persons and the management of their estates; and may appoint a committee or trustee for such persons, and make such orders and decrees respecting their persons and estates as to the court may seem proper. (Maryland Code, art. 16, § 96.)

Sale of Property of Non Compos Mentes.—Property may be sold on application of any creditor if the court is satisfied of the justice of the claim and there is no other means of paying the claim. The court may upon the application of the guardian decree the sale of the property, and order the money arising therefrom to be invested as the court may deem most advan-

tageous. On the death of such *non compos mentis* the principal sums arising from such sale shall descend to the persons to whom the property would have descended if the same had not been sold. The court may order real or leasehold property to be leased for any term of years, or may order the surrender of any lease. In all applications to sell the real or personal property of such person, if the court shall deem it for the interest and advantage of such person, it may decree a sale, lease, or surrender of such property on such terms and conditions as the court may prescribe. No sale, lease, or surrender shall be valid unless reported to and confirmed by the court. The court may allow to the committee or other person charged with the care of the person or estate of any *non compos mentis*, a sum not exceeding 10 per cent. of the income or expenditures for the care and trouble of such trustee or person. The court may decree that the property of such *non compos mentis* be sold for his support, or for the payment of expenses which the trustee may have incurred. (Idem, §§ 97–103.)

Confinement of Non Compos Mentes.—The court may, on the application of any trustee, and upon receiving proof that it is necessary and proper to confine such person, direct such trustee to send the person under his charge to any hospital in the vicinity of the city of Baltimore, provided he can be there received, to remain until a further order of the court. (Idem, § 104.)

Restoration.—When an application is made to have the commission superseded on the ground of recovery or restoration, the petitioner shall be entitled to have the question submitted to a jury impanelled under an order of court. (Laws of 1896, ch. 33.)

Inquisition as to Insane Pauper.—A county Circuit Court or the Criminal Court of Baltimore, when any person is alleged to be a lunatic or insane pauper, shall cause a jury of twelve men to be impanelled and charge the jury to inquire as to the sanity of such person, and if found insane, the court shall cause such person to be sent to the almshouse of the county or city to which he belongs, or to some other place best suited in the judgment of the court to his condition, there to be confined at the expense of the county or city until he shall have recovered and be discharged. (Maryland Code, art. 59, § 1.)

Confinement of Lunatic Pauper.—The county commissioners of any county may remove from the almshouse any lunatic pauper and cause him to be sent to the Maryland Hospital, and levy on the county the sum necessary to defray the expenses incidental to the removal of such lunatic and his maintenance in such hospital. Such expenses in no case to exceed the sum of one hundred and fifty dollars per annum. (Idem, § 2.)

Who Deemed a Lunatic Pauper.—No person shall be deemed a lunatic pauper who shall possess in his own right any property, real or personal, or be entitled to the use of any property by last will and testament, or deed of trust for his use or benefit. (Idem, § 3.)

Lunacy Commission.—A Lunacy Commission has supervision over all institutions in which insane persons are detained. (Idem, § 12.)

If, in its judgment, any person confined in any institution as insane be not insane, the commission may bring the matter to the attention of the State's attorney of any county, whose duty it shall be to apply to the proper tribunal for a writ of habeas corpus, to the end that proper inquiry and investigation may be had at once as to the mental condition of such person; and if the court shall be of the opinion that such person is not insane, then the court shall discharge such person; but if the court determines that such

person is insane, he shall be returned to the institution. Free access to all institutions shall be granted to the members of said commission, or their secretary; and the officers of such institutions shall furnish upon request all necessary information. The Lunacy Commission may issue compulsory process for the attendance of witnesses, administer oaths, and examine persons under oath. The managers of any such institutions shall have the right to appeal from the determination or action of said commission. (Idem, §§ 20–27.)

No asylum for the care or custody of the insane shall be kept without a license from said commission. (Idem, § 28.)

No person shall be committed to or confined as a patient in any institution, or almshouse, or other place for the care and custody of the insane or idiotic, except upon the written certificate of two qualified physicians, made within one week after separate examination by them of the alleged lunatic. No physician connected with any institution shall certify to the insanity of any person for the purpose of committing such person to such institution. (Idem, §§ 31, 32.)

Persons confined in such places shall be furnished with writing materials, and may correspond without restriction with some person chosen by themselves. (Idem, §§ 35, 36.)

Any person may voluntarily commit himself to an asylum for a period not exceeding three months. If at the end of that time he is unfit to be discharged, the Lunacy Commission shall be notified, and shall examine the case, and may authorize further treatment if deemed necessary. (Idem, § 37.)

MASSACHUSETTS.

(Boston Insane Hospital, New Dorchester; Worcester Insane Asylum, Worcester; Worcester Insane Hospital, Worcester; Danvers Insane Hospital, Hawthorne; Taunton Insane Hospital, Taunton; Northampton Insane Hospital, Northampton; State Hospital, Tewksbury; Asylum for Insane Criminals, State Farm; Westborough Insane Hospital, Westborough; State Colony for Insane, Gardner; Medfield Insane Asylum, Harding.)

Jurisdiction over the person and estate of insane persons is vested in the Probate Courts.

Guardians.—The Probate Courts may appoint a guardian of the person and estate of any insane person upon the application of the relatives or friends, or the mayor and aldermen or selectmen of the city or town, upon fourteen days' notice of the time and place appointed for the hearing, to be given to the alleged insane person. Guardians of spendthrifts and habitual drunkards may be appointed in the same way and upon the same notice by Probate Courts. The guardian of an insane person or spendthrift shall have the care and custody of the person of his ward, and the management of all his estate; he shall give bond in the manner prescribed for guardians of minors. A guardian may be discharged by the Probate Court on the application of the ward or otherwise, when it appears that the guardianship is no longer necessary. (Public Statutes of Massachusetts, ch. 139, §§ 7–12.)

Powers and Duties of Guardians.—Powers and duties of guardians for insane persons and habitual drunkards are the same as those prescribed for the guardians of minors. (Idem, §§ 29–34.)

Commitment of Insane Person.—A judge of the Supreme Court or of a Superior Court in any county, a judge of the Probate Court or of a police district or municipal court, may commit any insane person to either of the State lunatic hospitals. No person shall be committed without an order or

certificate signed by one of the judges. Such order or certificate shall state that the person is insane and is a fit person for treatment. The judge shall see and examine the person alleged to be insane, or state in his final order why it was not deemed necessary or advisable to do so. (Idem, ch. 87, §§ 11, 12.)

No person shall be committed without the certificate of two physicians certifying to the insanity of such person. Such physicians shall make oath that they are graduates of a legally chartered school or college, and that they have been in actual practice of their profession for at least three years. They shall have been duly registered as lunacy examiners and possess satisfactory standing, character, and professional knowledge. The certificate of lunacy shall bear date not exceeding ten days prior to commitment. The physicians shall have examined the alleged insane person within five days of signing the certificate. The form of the certificate is prescribed by statute. (Laws of 1895, ch. 286, 429.)

A person applying for the commitment of any lunatic to a State hospital shall first give notice in writing to the overseer of the poor of the place where the lunatic resides, or in the city of Boston to the Commissioners of Public Institutions, of his intention to make such application. (Laws of 1892, ch. 53.)

Application for the commitment of an insane person shall be accompanied by a statement containing facts as to the condition of the person alleged to be insane. The judge upon hearing the evidence may issue his warrant for the apprehension and bringing before him of the alleged lunatic, if, in his judgment, the condition or conduct of such person renders it necessary or proper to do so. (Public Statutes of Massachusetts, ch. 87.)

Inquisition.—The judge may summon a jury of six lawful men to hear and determine whether the alleged lunatic is insane. The jury shall be selected as the judge shall direct. The judge shall preside at such trial, and administer to the jury an oath faithfully and impartially to try the issue, and the verdict of the jury shall be final on the complaint. (Idem, §§ 17–22.)

Commission of Lunacy.—This consists of the State Board of Health, Lunacy, and Charity. It has power to investigate the question of the insanity and condition of any person committed to any lunatic hospital or asylum, public or private, and restrained of his liberty by reason of alleged insanity at any place within the Commonwealth, and shall discharge any person so committed or restrained, if, in its opinion, such person is not insane or can be cared for after such discharge without danger to others and with benefit to himself. (Idem, § 1.)

City Insane Asylums.—Cities of more than fifty thousand inhabitants may establish and maintain one or more asylums for the care and treatment of the chronic insane of such city and of any other city or town. Every such asylum shall be under the care of proper medical officers having experience in the care of the insane. The State Board shall visit and inspect every asylum so established, at least once in every six months, and may transfer the inmates thereof in the same manner as they now remove and transfer the inmates of other hospitals or asylums. (Laws of 1894, ch. 234.)

Care of Chronic Insane in Private Families.—The chronic insane of a quiet class may be boarded in families, if deemed expedient, by the State Board of Lunacy and Charity. The cost of boarding such insane person shall be paid from the appropriation for the support of State paupers in lunatic hospitals; but the rate paid shall not exceed the rate now paid in the

State lunatic hospitals. It shall be the duty of the board to visit at least once in three months all such insane persons boarded in families at the expense of the State; and all insane persons boarded at the expense of towns and cities, at least once in six months. (Laws of 1886, ch. 385.)

Commitment of Habitual Drunkards or Dipsomaniacs to State Lunatic Hospitals.—Any person suffering from dipsomania or habitual drunkenness may be committed to one of the State lunatic hospitals upon satisfactory evidence furnished to the judge before whom the proceedings for commitment are had, that such person is not of bad repute or of bad character apart from his habits of inebriety. Laws relative to persons committed on the ground of insanity apply to persons committed under the provisions of this Act. (Laws of 1885, ch. 339.)

Discharge of Inmates of Hospitals.—The board of trustees of any of the State lunatic hospitals may, by vote, confer on the superintendent of the hospital or asylum under their control, authority to discharge therefrom any inmate thereof committed as an insane person, upon written notice to the person who signed the petition for the commitment of such inmate. Said superintendent may, when advisable, permit any such inmate to leave the hospital for a period not exceeding sixty days, and receive him when returned without any further order of commitment. (Laws of 1883, ch. 78.)

Detention of Insane Persons without Treatment.—No overseer of the poor shall detain without remedial treatment any person whose insanity has continued for a period less than twelve months; all persons suffering from recent insanity shall have treatment in some hospital. When the State Board has reason to believe that any person is deprived of proper treatment and is confined in the almshouse or other place, whether such insane person is a public charge or otherwise, it shall cause application to be made to a judge for the commitment of such person to a hospital in the manner herein prescribed. (Laws of 1890, ch. 414.)

Transfer of Patients.—In transferring patients to and from institutions for the insane, in all cases where practicable trained nurses or attendants from such institutions shall be employed instead of officers of the law, and female nurses or attendants shall be employed to accompany female patients. (Laws of 1897, ch. 418.)

MICHIGAN.

(Michigan Asylum for Insane, Kalamazoo; Eastern Michigan Asylum, Pontiac; Northern Michigan Asylum, Traverse City; Asylum for Dangerous and Criminal Insane, Ionia; Upper Peninsula Hospital for the Insane, Newberry.)

The words "insane person" shall be construed to include an idiot, a *non compos*, lunatic, and distracted person. (Michigan Comp. Laws, § 50, subd. 7.)

Guardian.—Any judge of probate may appoint a guardian for an insane person or any person mentally incompetent to have the charge and management of his property, upon the application of relatives or friends of such person; or if such person is a county charge, upon the application of the directors of the poor. Notice shall be given such person not less than fourteen days before the time appointed for the hearing. If the judge finds that the person in question is incapable of taking care of himself and managing his property, he shall appoint a guardian. Pending the application for the appointment of a general guardian, a special guardian may be appointed, who shall hold office until the question of appointment of a general guardian be

decided, or until he shall be discharged by the judge of probate. (Idem, §§ 8709, 8710.)

Powers and Duties of Guardian.—Such guardian shall have the care and custody of the person and management of the estate of the ward. He shall give a bond to the judge of probate in the manner and form prescribed for the guardian of a minor. (Idem, § 8711.)

Guardian for Spendthrift.—When any person, by excessive drinking, gaming, idleness, or debauchery of any kind, shall so waste his estate as to expose himself or his family to want or suffering, or the county to charge or expense, any superintendent of the poor of the county, or director of the poor, or a justice of the peace, in which such spendthrift resides, may present a complaint to the judge of probate, setting forth the facts and circumstances of the case, and praying for the appointment of a guardian. Notice shall be given to such supposed spendthrift of the time and place of hearing, not less than fourteen days before the time appointed; but if, after a full hearing, it appears to the court that the facts of the complaint are true, he shall appoint a guardian of his person and estate. (Idem, §§ 8712, 8713.)

Powers and Duties of Guardian of Spendthrift.—Such guardian shall have the care and custody of the person and the management of the estate of the ward, and shall give a bond in the manner prescribed in respect to the guardian of an insane person. (Idem, § 8714.)

In General.—Every guardian, whether of an insane person or a spendthrift, shall be under the direction and control of the Probate Court, and shall have the same powers as to the estate of the ward as the guardian of a minor. (Idem, §§ 8717–8735.)

As to the appointment of guardians for drunkards and intemperate persons, see Laws of 1897, p. 151, Act 130 (Idem, §§ 8739–8743), where it is prescribed that the guardian may be appointed for such a person, upon the petition of the husband or wife, or some relative by blood, of the person for whom the guardian is asked, or by the supervisor of the town or alderman of the ward where, or one of the superintendents of the poor of the county in which such person resides. In such proceedings the probate judge may take the testimony of witnesses and examine the respondent, and shall determine whether such guardian should be appointed.

Confinement of the Insane.—When a person in indigent circumstances, and not a pauper, becomes insane, an application may be made to the judge of probate of the county where he resides, and the judge shall immediately notify such alleged insane person of the time and place of hearing, and shall summon the guardian and such relatives of the alleged insane person as are liable for his support. He shall call two legally qualified physicians and other credible witnesses, whose duty it shall be to attend and act in such case. Such judge shall fully investigate the facts, and, either with or without the verdict of the jury, determine the question of insanity and the question of his indigence. A jury shall be summoned when requested by the alleged insane person or a relative legally liable for his support, or by the prosecuting attorney for the county. If the judge certifies that such person is in indigent circumstances and his estate is insufficient to support him and his family, he shall be admitted into the asylum and supported there at the expense of the county to which he belongs. If it appear that the insane person has relatives legally liable for his support and who are able to contribute to such support, the probate judge shall order payment by such relatives of such sums as they may be able to pay. Provision is made for the collection of the amount ordered paid. If an insane person in in-

digent circumstances shall have been maintained by his friends in the asylum as a private patient for three months, and the superintendent shall certify that he is insane and requires further treatment, the judge may, without further evidence of the insanity, and if the indigence be established, make a certificate authorizing the admission of said patient into the asylum as a county charge. (Idem, § 1915, as amended by Laws of 1899, ch. 173, p. 255.)

Qualifications of Physician Certifying to Insanity.—The physician shall be of reputable character, a graduate of some incorporated medical college, a permanent resident of the State, registered according to law, not related to the alleged insane person nor to the person applying for the certificate, and shall have been in the actual practice of his profession for at least three years; such qualifications shall be certified to by the clerk of the county in which the physician resides. (Idem, § 1914.)

Admission of Private Patients to Asylums.—No private patient shall be admitted to any insane asylum except upon the certificate of two reputable physicians under oath, appointed by the judge of probate of the county where such person resides, to conduct an examination, and upon an order from said judge, setting forth that such person is insane and directing his removal to an asylum or institution for the care of the insane. (Idem, § 1913.)

Inquest.—The judge may institute an inquest and take proofs as to the alleged insanity before granting such order. He may in his discretion call a jury of six persons to determine the question of sanity. If satisfactory evidence is adduced showing the alleged insane person to be of unsound mind, he shall grant an order for the removal of such insane person to such institution, there to be supported as a private patient. (Idem, § 1913.)

Proceedings for the Commitment of Pauper Insane.—If any person being a pauper shall become insane, the county superintendent of the poor, or any supervisor of any city or town, may make application to the probate judge, who shall proceed to inquire into the question of the insanity of said person. He may call upon and compel the attendance of one or more legally qualified physicians and such other witnesses as he may deem necessary; and if satisfied of the insanity of such person, he shall make the same certificate and order for admission into the insane asylum as are provided in the case of persons in indigent circumstances. (Idem, § 1919.)

Maintenance of Insane.—The cost of the maintenance in the asylum of any indigent or pauper patient, received upon the order of any court or officer, shall be paid by the county from which he was sent to the asylum, except those termed "State patients." (Idem, § 1921.)

Asylums for the Insane.—Each asylum is controlled by a board of trustees. The medical superintendent, treasurer, steward, chaplain, assistant medical superintendent, and necessary assistant physicians, are appointed by the board. (Idem, §§ 1893, 1894, et seq.)

MINNESOTA.

(St. Peter State Hospital, St. Peter; Rochester State Hospital, Rochester; Fergus Falls State Hospital, Fergus Falls.)

Commitment of Insane Persons.—A warrant may issue from the Probate Court of any county, upon information showing that there is an insane person in the county needing care and treatment, and that it is dangerous for him to be at large, to apprehend such person. Upon the filing of

such information the court shall make an order directed to two reputable persons, one of whom shall be a duly qualified physician, and such persons with the judge shall constitute a jury to examine the person alleged to be insane, and they shall ascertain the facts of sanity or insanity. Each of such persons shall be sworn to examine the patient impartially and to the best of his ability. The Probate Court may summon such witnesses as are necessary. Certain questions prescribed by statute shall be asked in the examination. Upon the completion of such examination the jury shall report their findings in writing. Such findings shall be that the person is sane or insane. The relatives or friends of any person alleged to be insane or found insane shall have the right to the charge and care of such person, upon the giving of a bond for the proper care and safekeeping of such person. If such person is found to be insane, he may be committed by order of the judge to one of the hospitals for the insane. If the person committed is a female, she shall be accompanied to the hospital by a woman or by her husband. (Probate Code, §§ 266–275.)

Guardians of Incompetents.—A Probate Court may appoint a guardian or guardians of any person who, by reason of old age or loss or imperfection of mental faculties, is incompetent to have the charge or management of his property, or a person who, by excessive drinking, gaming, idleness, or debauchery, so spends or wastes his estate as to be likely to expose himself or his family to want or suffering, either upon the application of the county commissioners of the county where such person resides or upon the petition of any relative or friend of such person; which petition shall set forth the facts and be verified by the affidavit of the petitioner to the effect that he believes the facts as so stated are true. The petitioner, or any person interested, may file a copy of the petition and notice and proof of service in the office of the register of deeds; and if a guardian shall be appointed in the proceedings, all contracts made by the alleged incompetent after such filing are void. Upon the presentation of the application, the Probate Court shall fix a time for a hearing, and shall cause notice to be given to the person proposed to be put under guardianship at least fourteen days prior to the time fixed for the hearing. All competent evidence shall be considered at the hearing, and if it appear that the person is such an incompetent, the court shall appoint a guardian or guardians of his person and estate. (Idem, §§ 142–144, as amended by Laws of 1893, ch. 116, § 2.)

Powers and Duties of Guardian.—Every guardian so appointed shall have the care and custody of the person of his ward and the management of all his estate until such guardian is discharged. The provisions relating to the powers and duties of the guardians of minors, as prescribed in the Probate Code, § 1477 ff., apply to guardians of incompetents.

Restoration to Capacity.—The fact of the restoration of an incompetent person shall be judicially determined upon the application of such insane person or his guardian, relatives, or friends. Notice shall be given of a hearing to the guardian of the person. On the hearing the guardian, relative, or friend may contest the right to the relief demanded. Witnesses may be summoned and examined by the court of its own motion. If it be found that the person is of sound mind and capable of taking care of himself and property, his restoration to capacity shall be adjudged and the guardianship shall cease. (Idem, § 146.)

Hospitals for the Insane and Commitment Thereto.—The State hospitals are under the charge of a board of trustees, composed of five members appointed by the governor, by and with the advice and consent of the

senate. Trustees have the general control of the hospitals, and may make all by-laws necessary for the government of the same, appoint for each hospital a medical superintendent and an assistant medical superintendent, fix all the salaries not otherwise determined by law, and remove all officers appointed by them, except the superintendent, who shall only be removed for good cause shown, and then only with the approval of the governor. The superintendent of each such hospital shall have the control and management of the hospital, and may employ and discharge all attendants, servants, and employés at his pleasure, and suspend any subordinate officer until an examination is had before the board of trustees, and immediately upon such suspension he shall report the fact to said board. He shall give immediate notice to the next of kin of each patient under his charge of the death, serious illness, or any special change in the condition of such patient, and answer promptly and fully all letters of inquiry received from the relative of any patient in said hospital. (Laws of 1893, ch. 5, §§ 1-13.)

Commitment; Examination.—When information in writing is received by the probate judge of any county that there is an insane person in his county needing care and treatment, he shall issue his order directing the sheriff or some other suitable person to bring before him the person alleged to be insane for the purpose of examination; at the same time he shall appoint a jury composed of two examiners in lunacy, who, with the judge, shall comprise such jury. The county attorney, or an attorney appointed by him, shall appear and protect the interests of the alleged insane person. If the examiners' jury find the information to be true, they shall certify such fact to the judge within twenty-four hours, and the judge shall thereupon commit the insane person to the proper State hospital or a private asylum. (Laws of 1895, ch. 119, §§ 3, 4.)

Arrests of Insane Persons.—No alleged insane person shall be arrested and committed to jail unless he has committed some crime or is dangerous or disorderly, or there are reasonable grounds to believe that he will do injury to himself or others or to property. (Laws of 1893, ch. 5.)

Privileges of Insane in Hospital.—Every inmate committed to any hospital for the insane, upon entering the institution, may choose as a correspondent an individual not connected with the institution, with whom he shall be allowed to communicate freely in writing. Each inmate may choose a new correspondent every three months. The superintendent shall keep registered and posted in some public place at the institution the name and post-office address of each correspondent and the name of the inmate choosing such correspondent. When any person is chosen as a correspondent, the superintendent shall notify him within three days that he has been chosen, and shall inquire whether he will act. Each inmate of any hospital may communicate in writing with the governor and the secretary of the board of trustees, in the same manner as with the correspondent. Any person refusing or neglecting to allow any such personal privilege shall be deemed guilty of a misdemeanor. (Laws of 1893, ch. 5, §§ 28, 29-33, 34.)

MISSISSIPPI.

(State Insane Hospital, Asylum P. O.; East Mississippi Insane Asylum, Meridian.)

Writ de Lunatico Inquirendo.—The Chancery Courts have jurisdiction of writs of lunacy, to be exercised by the clerks at any time subject to the approval of the court. Any relative of a lunatic or insane person may procure him to be so adjudged. If the relatives or friends of any insane

person allow him to go at large, the clerk of the Chancery Court may, upon application of any citizen, direct the sheriff by writ of lunacy to summon the alleged lunatic or insane person to contest the application, and six freeholders to make inquiry thereof. If the person shall be adjudged insane by the jury, the clerk shall direct the sheriff by writ to arrest him and place him in one of the asylums, if there be a vacancy; and if not, confine him in the county jail until there be room in the asylum. If the person be adjudged harmless and indigent and not in need of special treatment, he shall be sent to the poorhouse. (Mississippi Code, §§ 2835–2837.)

Admission into Asylums.—The superintendent of each asylum shall admit and receive therein all persons ordered to be confined therein in the order of application, if there be a vacancy in the asylum. The expenses of the inquiry and of the removal to and from the asylum shall be borne by the estate of the lunatic, if he have any; and if not, by the person required by the pauper laws to support him; but in the first instance the expenses are to be paid by the county. (Idem, §§ 2838–2840.)

Lunatic Asylums.—The control and management of the asylums for the insane are vested in a board of five trustees, appointed by the governor, with the advice and consent of the senate, who have charge of the interests of the asylum, and manage and direct its affairs, and make all proper by-laws and regulations for its control and government. The trustees are required to make regular and frequent inspection of the asylums, for which purpose one or more of them shall visit the asylums at least once in every month. Superintendents of the asylums are appointed by the governor, and are required to be skilful physicians. The superintendent has the supervision of the buildings, with their furniture, fixtures, and stock, and the direction and control of all persons and officers therein. The white and colored races are kept separate in the asylums. In no case shall discipline be enforced to the extent of corporeal punishment. (Idem, §§ 2807–2817, 2820, as amended by Laws of 1898, ch. 67.)

Guardians.—The Chancery Court may appoint guardians of persons adjudged, upon inquisition, to be of unsound mind, upon its own motion or on the application of a relative or friend or of a member of the board of supervisors. If the person has not been adjudged insane, the writ *de lunatico inquirendo* shall issue upon any such application, and if upon such inquisition the person be adjudged of unsound mind and incapable of taking care of himself or property, the court may appoint a guardian. The Chancery Court may also appoint guardians for drunkards and opium-eaters on the application of a relative or friend. In such case the court shall examine into the question and determine whether the person be an habitual drunkard or opium- or morphine-eater, and for that purpose may summon and hear witnesses and hear the parties and their evidence; and if the court be satisfied that the person is an habitual drunkard, opium- or morphine-eater, it shall appoint a guardian of his person and estate. The Court of Chancery may direct the confinement of any person adjudged an habitual drunkard or an habitual opium- or morphine-eater in an asylum. (Idem, §§ 2212–2215, 2217.)

Powers and Duties of Guardians.—Guardians shall make an inventory of the estate and account to the court as often and in the same manner as executors and administrators. Guardians shall improve the estate committed to their charge, and apply so much of the income as may be necessary to the comfortable maintenance and support of the ward and his household or family. (Idem, §§ 2219, 2220, as amended by Laws of 1896, ch. 97.)

MISSOURI.

(State Hospital, No. 1, Fulton; State Hospital, No. 2, St. Joseph; State Hospital, No. 3, Nevada; State Hospital, Farmington; City Asylum, St. Louis.)

Inquiry as to Insanity.—If information in writing be given to the Probate Court that any person in its county is an idiot, lunatic, or person of unsound mind, and incapable of managing his affairs, and praying that an inquiry thereinto be had, the court, if satisfied that there is good cause for the exercise of its jurisdiction, shall cause the facts to be inquired into by a jury. The alleged insane person shall be notified of the proceeding, unless such person is ordered to be brought before the court. Any judge of the county court, or justice of the peace, sheriff, coroner, or constable, may make application to the Probate Court for the exercise of its jurisdiction. (Statutes of Missouri, §§ 5513–5516.)

Appointment of Guardian.—If the person be found insane by the jury, the court shall appoint a guardian of his person and estate. Every guardian so appointed is required to give a bond for the due and proper care of such person and the management of his estate to the best advantage. Every such guardian shall take charge of the person and provide for his support and maintenance. He shall collect and take into his possession all the personal property, and within sixty days after his appointment file a just and true inventory of the real and personal estate of his ward. An additional inventory may be required from time to time whenever any property belonging to such estate shall be discovered, and all such inventories shall be attested and verified. The real estate may be sold on the petition of the guardian when the personal property is insufficient for the discharge of the debts and the maintenance of the ward and his family. Every such sale shall be made under the direction of the Probate Court. (Idem, §§ 5517–5523.)

Claims against Insane Person.—All demands against the estate of an insane person are required to be presented to the Probate Court on notice to the guardian. All demands not presented within two years from the appointment of the guardian are barred. The guardian is required to publish for three weeks that letters of guardianship have been granted to him, and requesting the presentation of claims. (Idem, §§ 5529*a*–5529*c*, as amended by Laws of 1899, p. 227.)

Confinement of the Insane.—If any person shall be furiously mad, or so far disordered in his mind as to endanger his own person or the person or property of others, it shall be the duty of the guardian or other person under whose care he may be, and who is bound to provide for his support, to confine him until the next sitting of the Probate Court, which shall make such order for the restraint, support, and safekeeping of such person as the circumstances of the case may require. If such person shall not be confined by the person having charge of him, or if there be no person having such charge, any judge of a court of record or any two justices of the peace may cause such insane person to be apprehended. If any insane person be admitted to any State lunatic asylum as a patient, the guardian shall pay for his support and expense at such asylum out of the estate of such ward. If such insane person comes under the class of insane poor persons, such person shall be supported and maintained by the county in the manner provided by law. (Idem, §§ 5554–5557.)

Insane Asylums.—The superintendent of each asylum shall be a physician of knowledge, skill, and ability in his profession and of experience in

the management and treatment of the insane. He shall not while superintendent engage in the practice of his profession, but shall devote himself exclusively to the supervision and care of the asylum and its inmates. He shall be the chief executive officer of the asylum, and have the care and control of everything connected therewith. (Idem, §§ 471, 472.)

Admission into Asylum.—Persons afflicted with any form of insanity may be admitted into the asylum when the superintendent deems it probable that their condition can be improved, and any patient may be discharged by the superintendent whenever he may believe that the condition of such patient cannot be improved by a longer stay in the asylum. Pay patients not sent to the asylum by order of the court may be admitted in accordance with the statutes and the by-laws of the asylum. The several county courts may send to the asylum such of their insane poor as may be entitled to admission thereto. The counties thus sending shall pay semi-annually in cash, in advance, such sums for the support and maintenance of their insane poor as the board of managers may deem necessary. The indigent insane of the State shall have preference over those who have the ability to pay for their support in the asylum; and if there be no provision in the asylum for the accommodation of all the insane persons in the State, recent cases of insanity—meaning cases of less than one year's standing—shall have preference over cases of more than one year's standing. (Idem, §§ 473–486.)

MONTANA.

(Montana Insane Asylum at Warm Springs.)

Appointment of Guardian.—Upon the verified petition of a relative or friend that the person is insane or mentally incompetent to manage his property, the District Court, or a judge thereof, shall give notice to the alleged incompetent person of the time and place of hearing the case, not less than five days before the time so appointed, and such person, if able, shall be in attendance before him on the hearing. If, after a full hearing and examination, it appears to the judge that the person is incapable of taking care of himself and managing his property, he shall appoint a guardian of his person and estate. Every such guardian has the care and custody of the person of his ward, and the management of all his estate, until he is legally discharged. He shall give a bond to such ward in the manner prescribed for the guardian of a minor. Every such guardian has all the powers and duties of guardians of minors, as specified in Chapter XIV., of Title XII. of the Code of Civil Procedure (§§ 2970–2972. III. 39).

Restoration of Insane Person.—Any person declared insane, or his guardian or any relative within the third degree, or any friend, may petition the District Court to have the fact of his restoration to capacity judicially determined. Upon receiving the petition the judge shall appoint a day for the hearing, and upon the petitioner's request order an investigation before a jury, which shall be summoned and impanelled in the same manner as other juries in civil actions. A notice shall be given to the guardian, husband, or wife, if there be one, or father or mother if living in the county. On the trial any person, in the discretion of the judge, may be subpenaed and examined as in other cases. If it be found that the insane person be of sound mind and capable of taking care of himself and his property, his restoration shall be adjudged and the guardianship shall cease. (Idem, § 2973.)

Commissioners of the Insane.—The governor, secretary of State, and the attorney-general constitute the State Board of Commissioners for the

Insane. Such commissioners have power to prescribe rules for their own government; and to provide by contract for the care, custody, maintenance, and treatment of the insane in a safe and suitable building for that purpose, which is known as the State Insane Asylum. The contract therefor shall be let to the lowest and best bidder; and such contract shall require the person entering into the same to receive all persons adjudged insane, and to keep, maintain, and treat them in accordance with the requirements of the commissioners. Such contract shall not continue for more than two years, nor beyond the date of completion of the State Asylum for the Insane. Such contract shall require the person entering into the same to furnish all necessary attendance and assistants required by the medical and all other departments in such asylum, and that there be furnished well-ventilated, properly warmed, and suitable rooms or apartments, and good and wholesome food and clothing for the patients. (Political Code, §§ 2261–2269.)

General Provisions.—The board may send a patient to an asylum out of the State, at the expense of the State, if the patient is indigent. Idiotic persons are considered as insane. The board may discharge persons fit to be at large. Patients shall be permitted to write to persons selected by them. (Idem, §§ 2280–2289.)

Examination and Committal of Insane Persons.—If it appears to a magistrate that a person is so far disordered in his mind as to endanger health, person, or property, he shall issue a warrant directing such person to be taken before a district judge in the county, or in his absence before the chairman of the county commissioners. Witnesses shall be subpœnaed, including at least two graduates of medicine. The physicians shall hear the testimony and make a personal examination of the alleged insane person. The physicians shall thereupon make a certificate describing the patient and his condition, in a form prescribed by statute. The judge, if he believes the person insane, shall order his commitment, and direct the sheriff to deliver him, with the order of the judge and the certificate of the physicians, to the officer in charge of the insane asylum. (Idem, §§ 2300–2308, as amended by Laws of 1897, p. 163.)

NEBRASKA.

(Nebraska Hospital for the Insane, Lincoln; Asylum for the Chronic Insane, Hastings; Norfolk Hospital for the Insane, Norfolk.)

The term "insane" includes every species of insanity or mental derangement. The term "idiot" is restricted to persons supposed to be naturally without mind. No idiots shall be admitted as patients to a State hospital. (Nebraska Comp. Statutes, § 3372.)

Guardians.—The Probate Court may appoint a guardian for an insane or incompetent person upon the application of relatives or friends of such person, and after notice given to the supposed insane person of the time and place of hearing, not less than fourteen days. If, after a hearing, the court determines that such person is incapable of managing his property or taking care of himself, he shall appoint a guardian. Such guardian shall have the care and custody of the person and the management of all of the estate of his ward. Upon a like application a guardian may be appointed for a spendthrift after due notice and a full hearing. The powers and duties of guardians as to the management of the estates of their wards are similar in all respects to those of guardians of minors. (Idem, §§ 3225–3227.)

Insane Asylums.—The insane asylum is under the charge of three

trustees, who have the general control and management of the hospital, with full power to make all by-laws necessary for their government. It is the duty of a majority of the board to visit the hospital quarterly. The governor shall appoint a superintendent, who may appoint two assistant physicians for the hospital for the insane, one of whom shall be a woman. The superintendent shall be a physician of acknowledged skill and ability, and a graduate of a regular medical college. He is the chief executive officer of the hospital, and holds his office for a term of six years. He has the entire control of the medical, moral, and dietetic treatment of the patients, and shall see that the several officers of the institution faithfully and diligently discharge their respective duties. (Idem, §§ 3321, 3325–3331.)

There are also a State hospital for the insane at Norfolk, and one for the chronic insane at Hastings, both of which are under the management of the Board of Public Lands and Buildings. The governor appoints the superintendents and other officers of these asylums. (Idem, §§ 3377–3379.)

County Commissioners of Insanity.—There is in each county a board of commissioners, consisting of three persons, styled the Commissioners of Insanity. The clerk of the District Court is ex-officio a member of the board and the clerk of the same. The other members are appointed by the judge of the District Court, one of whom shall be a respectable practising physician and the other a respectable practising lawyer. Such commissioners have cognizance of all applications for admission into the State hospital or for the safekeeping otherwise of insane persons within their respective counties. (Idem, §§ 3336–3339.)

Admission into the Hospital.—Application for admission to the State Hospital shall be made in writing in the nature of an information verified by affidavit, stating that the person in whose behalf the application is made is believed by the informant to be insane and a fit subject for custody and treatment in the hospital. On the filing of such information the Commissioners of Insanity shall investigate the grounds of the information. For this purpose they may require the person alleged to be insane to be brought before them, and they shall provide him with suitable custody until their investigation shall be concluded. They shall hear the testimony for and against such application, if any is offered. The commissioners shall appoint some regular practising physician of the county to visit or see such person and make a personal examination touching the truth of the allegations in the information. Such physician shall certify that he has in pursuance of his appointment made a careful personal examination, and on such examination he finds the person in question insane, if such be the fact. On the return of the physician's certificate the commissioners shall conclude their investigation and, having done so, shall find whether the person alleged to be insane is insane, and if so, whether he is a fit subject for treatment and custody in the hospital. If they find such person insane and a fit subject for custody and treatment in the hospital, they shall issue their warrant stating such finding, authorizing the superintendent of the hospital to receive such person as a patient therein. Such warrant and duplicate, with finding and certificate of the physician, shall be delivered to the sheriff of the county, who shall execute the same by conveying such person to the hospital and delivering him with such duplicate and physician's certificate and finding to the superintendent. (Idem, §§ 3340–3342.)

NEVADA.

(Nevada Hospital for Mental Diseases, Reno.)

Jurisdiction.—The District Courts have jurisdiction over the person and estates of idiots and insane persons. (Statutes of Nevada, § 2439.)

Inquisition as to Insanity.—Upon an application under oath, setting forth that a person by reason of insanity is dangerous to be at large, the district judge shall cause such person to be brought before him, and shall summon at the same time and place two or more witnesses having had frequent intercourse with the person during the time of his alleged insanity, who shall testify as to their knowledge of such person. At the same time he shall cause two graduates of medicine to appear and examine the person, and if after such examination and a careful hearing they certify on oath that the person is insane, and if the district judge be satisfied of the existence of insanity and that it would be dangerous for such insane person to be at large, he shall direct the sheriff to convey such insane person to the State capitol and place him under charge of the secretary of State. (Idem, § 1457.)

Appointment of Guardian.—When the insane person is able by the possession of property to pay the expenses attendant upon his commitment and maintenance at the State Asylum, the judge shall appoint a guardian, who shall be subject to the general law in relation to guardians as far as the same may be applicable. If there is not sufficient money in hand, the judge shall order the sale of the property of such person, or so much thereof as may be necessary, and the guardian shall appoint trustees to pay all proper costs and charges incidental to the care and support of such insane person. If such insane person has no property, but has relatives in the degree of husband or wife or father or mother, of sufficient means to support such insane person, the judge shall order all such expenses to be paid by them, and may assess the same among such kindred as he may deem just and equitable. (Idem, § 1458.)

Commissioners for the Care and Maintenance of Indigent Insane.—The governor, State comptroller, and State treasurer constitute a board of commissioners for the purpose of providing for the care and maintenance of the indigent insane. (Laws of 1887, ch. 33.)

Such board has full power and control of the State asylum, and may establish such rules and regulations for the care thereof as they may deem proper. They shall elect one resident physician, who shall be general superintendent, subject to the order and direction of such board. (Statutes of Nevada, §§ 1451, 1452.)

Fund for Indigent Insane.—A fund shall be established, to be known as the Indigent Discharged Patients' Fund, to be applied to the relief of indigent patients discharged from the Nevada Hospital for Mental Diseases, under the direction of the commissioners for the care of indigent insane. (Laws of 1899, ch. 123.)

NEW HAMPSHIRE.

(New Hampshire State Hospital, Concord.)

The words "insane person" shall include every idiot, *non compos*, lunatic, insane or distracted person.

The word "spendthrift" shall include every one liable to be put under guardianship on account of excessive drinking, gaming, idleness, debauchery, or vicious habits of any kind. (Statutes of New Hampshire, title 2, ch. 2, §§ 18, 19.)

Guardians of Insane Persons and Spendthrifts; Appointment.—On the application of a relative or friend of an insane person, or of the overseer of the poor of the town where he lives, the probate judge shall cause inquisition with notice to be made by three suitable persons. If, upon the return of the inquisition and due examination, it is decreed that the person is insane, the judge shall appoint a guardian over him. Any person who by excessive drinking, etc., so wastes his estate or neglects his business as to expose him or his family to want or suffering, shall be deemed a spendthrift; and upon complaint in writing made to the judge of probate the judge shall appoint a day of hearing; and if upon due notice and examination it appears that such person is a spendthrift, the judge shall appoint a suitable person to be his guardian. Every guardian so appointed shall immediately give public notice thereof in some newspaper circulated in the vicinity, or in such newspaper as the court shall direct. (Idem, title 24, ch. 179.)

Powers of Guardians.—The guardians of a spendthrift may employ him or his children in any suitable labor, or bind them out by written contract for a term not exceeding one year. If the judge is satisfied that the estate of the ward is not sufficient to discharge his just debts, he may decree that the estate be settled as insolvent, and thereupon such proceedings may be had as in the case of insolvent estates of deceased persons. Such guardians possess all the powers of guardians of minors, and, subject to the direction of the probate judge, shall have the management and control of the property of the ward.

Commitment to Asylum.—The parent, guardian, or friends of any insane person may cause him to be committed to an asylum with the consent of the trustees, and there supported on such terms as they may agree. An insane pauper supported by the town may be committed to an asylum by order of the overseers of the poor, and there supported at the expense of the town. If the overseers neglect to make such order, the Supreme Court or any judge thereof may order such pauper to be committed. If any insane person is in such condition as to render it dangerous for him to be at large, any judge of probate on the petition of any person, and upon notice to a selectman of his town or to his guardian, may commit such insane person. No person shall be committed to the asylum except by an order of the Supreme Court or any judge of probate, without the certificate of two reputable physicians that such person is insane, given after a personal examination made within one week of the committal. (Idem, title 3, ch. 10, §§ 14–19.)

When application is made for the committal of any person to the asylum for the insane, the court or judge may appoint two reputable physicians to examine said person, with or without notice to him or her. Said physicians shall immediately report the result of their examination to said court or judge, who may, upon such report and such evidence as can be produced, order said person to be committed to said asylum, when there is sufficient reason for making such order. (Laws of 1895, ch. 14.)

Support at Asylum.—Any insane person thus committed who has no means of support and no relatives of sufficient ability chargeable therewith, and no settlement in any town of this State, shall be supported by the county from which he was committed. Insane persons charged with crime, the punishment for which is death or confinement in the State prison, shall be supported at the expense of the State. The county or town paying the expenses of the support of an inmate may recover the amount paid of the inmate if of sufficient ability to pay. (Statutes of New Hampshire, §§ 20–25.)

Discharge from Asylum.—Any person may be discharged from such asylum by any three of the trustees, by the commission of lunacy, or by a justice of the Supreme Court, whenever a further detention at the asylum is in their opinion unnecessary. (Idem, § 27.)

Trustees to Visit Asylum.—One of the trustees, at least twice every month and without previous notice, shall visit the asylum and give opportunity to every inmate to make to him in private any statements he may wish. If, in their judgment, a further detention is unnecessary, it shall be their duty to discharge such inmate. (Idem, § 28.)

Commission of Lunacy.—All persons deprived of their liberty by being committed to custody as insane persons shall be wards of the State and subject to State supervision. The State Board of Health shall constitute a commission of lunacy. The commission, or one or more of its members, shall, without previous notice, visit and thoroughly inspect all institutions for insane persons as often as once in four months. The commission shall keep a correct record of the number of commitments, discharges, and deaths at each asylum, and of the age, sex, and nationality of each person committed, discharged, or deceased, and report the same annually to the governor and counsel. The superintendent of every asylum shall, within three days after the commitment thereof of any person, notify the commission, and such superintendent shall at all times furnish to the commission such information as it may request. (Idem, §§ 31–35.)

NEW JERSEY.

(New Jersey State Hospital, Trenton; New Jersey State Hospital, Morris Plains.)

Inquest of Idiocy and Lunacy.—All cases of idiocy and lunacy are determined by inquest and a commission of idiocy and lunacy issued out of the Court of Chancery and returnable thereto. If, upon such inquest, idiocy or lunacy be found, the chancellor shall cause to be transmitted to the Orphans' Court of the county where such lunatic or idiot resides a certified copy of all proceedings. (General Statutes of New Jersey, p. 1696.)

Appointment of Guardian.—Upon return made and application therefor to the Orphans' Court, such court shall appoint a guardian of such idiot or lunatic, who shall have the care and safekeeping of his person and property. (Idem, pp. 1696, 1697, §§ 1–3.)

Court may Order Sale of Lands.—If any such idiot or lunatic is justly indebted beyond his ability to pay, or if his personal estate is insufficient for the support and maintenance of such lunatic or idiot and his household, the Orphans' Court of the county in which the property is situated or the chancellor may order the guardian to sell such part of the lunatic's real property as the court may deem sufficient to pay such debts and the necessary expense for the support and maintenance of the idiot or lunatic or his household. The guardian shall make a deed to the purchaser for the real estate so sold, which shall set forth the order at large, and shall vest in the purchaser as good a title to the estate as the idiot or lunatic possessed at the time of the making of the order. Such lands may also be sold by order of the chancellor upon an application by the guardian. (Idem, pp. 1697, 1698, 1703.)

Bond of Guardian.—Every guardian ordered to sell lands shall, before or at the time of making the report of the sale, enter into a bond in such security as the chancellor shall deem sufficient for the faithful discharge of the trust committed to him. (Idem, p. 1697, § 10.)

Proceeds of Sale.—All moneys arising from any sale shall be put out at interest on good and sufficient security of unencumbered real estate; or if the chancellor shall so direct, in bonds of United States or of this State, but in no other way whatever. Whenever after a sale so made it shall become necessary to apply any of the proceeds to the support of such idiot or lunatic, the guardian shall apply to the Orphans' Court, which shall, on due proof in addition to the oath of such guardian that such application is necessary, order and direct the appropriation of so much of such proceeds as is necessary for such support, specifying in its order the amount per year to be applied for such support. (Idem, p. 1698.)

New Sureties to Bond.—Whenever the Orphans' Court shall suspect that the sureties of the guardian or any of them are failing or in dubious circumstances, they may require such guardian to give additional sureties, and upon refusal or neglect may displace such guardian and appoint another in his place. (Idem, p. 1699.)

Death of Guardian.—In case of the death of any guardian, the Orphans' Court shall forthwith appoint another. Whenever a new guardian is appointed, the representatives of the deceased guardian shall account to such new guardian for all property in their possession or under their control belonging to such idiot or lunatic. (Idem, 1877, p. 1699.)

Account of Guardians.—Guardians of idiots or lunatics shall, once in three years or oftener, in case the Orphans' Court shall so order and direct, render to the court from which the appointment was received a true account of the administration of the estate of such idiot or lunatic, and on the death of such idiot or lunatic, or upon his restoration, the guardian may be compelled to render an account of his administration of the estate in the same manner as executors and administrators. (Idem, p. 1699.)

Idiot or Lunatic not to be Imprisoned.—No lunatic or idiot during the time of his lunacy shall be committed or detained in prison for want of bail or his body in execution in any civil action; and in case any idiot or lunatic shall be so arrested in any civil suit, he shall be discharged on motion by the court out of which the process issued, or upon a writ of habeas corpus issued out of the Court of Chancery or the Supreme Court. (Idem, p. 1700.)

Dangerous Lunatic at Large.—Any two justices of the peace of any county in which any lunatic furiously mad or dangerous shall be found, may, by warrant, cause such person to be apprehended and kept safely locked up, and chained if necessary, in any place provided in such county for the reception of maniacs or lunatic persons; and if there be no such place, in the jail of such county. If such lunatic be possessed of property, the expense incurred by such detention shall be paid therefrom; but if he possess no property, then such expense shall be charged upon the city or township in which such person is legally settled in the same manner as other poor persons. (Idem, p. 1700; and see ch. 172 of the Laws of 1888, as amended by ch. 44 of the Laws of 1891, and Laws of 1894, p. 283.)

State Hospitals.—The State hospitals for the insane are located at Morris Plains and at Trenton. The management and control of each hospital are in a board of managers, consisting of eight persons appointed by the governor, by and with the advice of the senate, and holding office for the term of five years. The State is divided into districts, from each of which patients are committed to the hospital situated therein. Each board is required to make a visit to each county asylum in its district at least once in each year. (Laws of 1897, ch. 205, p. 447.)

Boards of managers are authorized to make, adopt, and enforce rules and

regulations for the distribution between the two hospitals of all patients sent thereto, and for the removal of patients from one to the other. They also have power to appoint medical directors for each hospital, and as many assistant physicians as they shall deem necessary. Medical directors have charge, direction, and control of all patients and of all persons engaged in the care of such patients, subject to the rules, regulations, and by-laws adopted by the board. Such managers shall maintain an effective inspection of such hospitals, for which purpose one or more of them shall visit each of them at least once in every week, two or more at least once in every month, a majority at least once in every three months, and the whole board once a year. They shall annually report to the governor the year's operations and the actual state of the hospitals. (General Statutes of New Jersey, p. 1982.)

Commitment of Insane Persons to Asylums.—No person shall be admitted to a hospital for the insane either public or private, except on the filing of a request for the admission of such person, by his relatives or some other persons interested in his welfare, with the medical director of the hospital; there must also accompany the request a certificate under oath, of two physicians, setting forth such person's insanity. No person shall be so confined for longer than fifteen days unless the approval of a justice of the Supreme Court or a judge of the Circuit Court or Court of Common Pleas be indorsed on the certificate of insanity. Such justice or judge may institute an inquiry, and may call a jury to ascertain the question of insanity. If the justice or judge making the inquiry determine that the person is not insane, he shall so certify to the medical director, who shall thereupon discharge the patient. (Laws of 1898, ch. 130, § 1.)

Qualifications of Medical Examiners; Certificates.—Each physician certifying as to the insanity of a person shall be of reputable character, a graduate of a legally incorporated medical college, a permanent resident of the State, and of five years' experience in the practice of his profession. Each certificate shall be made on a personal examination of the patient, according to forms prescribed by the managers of the asylum, shall be dated not more than ten days prior to the commitment, and shall contain a thorough description of the patient. (Idem, § 2.)

Commitment of Indigent Insane.—If the patient is indigent, the justice or judge in conducting the inquiry shall ascertain and state in his certificate whether the patient has sufficient estate to support himself, or his family, if any. If he is indigent, the fact shall be reported to the county freeholders, who are required to raise the money requisite for the support of the patient. (Idem, § 5.)

NEW YORK.

(Manhattan State Hospital, East, Ward's Island, New York City; Manhattan State Hospital, West, Ward's Island, New York City; Manhattan State Hospital at Central Islip, L. I.; Long Island State Hospital, Flatbush, Brooklyn; Long Island State Hospital, King's Park; State Homeopathic Hospital, Middletown; Hudson River State Hospital, Poughkeepsie; Utica State Hospital, Utica; Binghamton State Hospital, Binghamton; St. Lawrence State Hospital, Ogdensburg; Matteawan State Hospital (for criminal insane), Fishkill Landing; Willard State Hospital, Willard; Rochester State Hospital, Rochester; Buffalo State Hospital, Buffalo; Gowanda State Homeopathic Hospital, Gowanda; Dannemora State Hospital (for insane convicts), Dannemora.)

Jurisdiction.—The jurisdiction of the Supreme Court extends to the custody of the person and the care of the property of a person incompetent to manage himself or his affairs in consequence of lunacy, idiocy, habitual

drunkenness, or imbecility arising from old age or loss of memory and understanding, or other cause. The court shall preserve the property of the incompetent person from waste or destruction, provide for the payment of his debts and for his safekeeping, and the maintenance and education of his family out of the proceeds of his estate. This jurisdiction is exercised by means of a committee of the person or a committee of the property. (Code of Civil Procedure, §§ 2320–2322.)

Appointment of Committee.—An application for the appointment of a committee must be made by petition, which may be presented by any person. Where the incompetent person has property which may be endangered in consequence of his incompetency, and no relative or other person applies for the appointment of a committee, the overseer or superintendent of the poor of the town, district, county, or city in which he resides shall apply to the proper court for the appointment of such a committee. The petition shall be in writing and verified by the affidavit of the petitioner to the effect that the matters of fact therein stated are true. If the court is satisfied that a committee ought to be appointed, the court shall make an order directing that the commission issue to one or more fit persons, or that the question of fact arising upon the competency of the person be tried by a jury. The commission shall direct the commissioners to cause the sheriff to procure a jury, and that they inquire by the jury into the matters set forth in the petition, and also into the value of the real and personal property of the person alleged to be incompetent and the amount of his income. The commissioners or a majority of them shall issue a precept to the sheriff, requiring him to summon not less than twelve nor more than twenty-four indifferent persons to appear before the commissioners at the specified time and place, to make inquiry as commanded by the commission. All the commissioners shall attend and preside at the hearing; at least twelve jurors shall concur in the finding. If twelve do not concur, the jurors shall report their disagreement to the commissioners, who shall thereupon discharge them and issue a new precept to the sheriff to procure another jury. If the order directs the trial by a jury at a trial term, the order shall state distinctly and plainly the questions of fact to be tried, which may be settled as where an order for a similar trial is made in an action. Upon the return of the commission with the inquisition taken thereunder, or the rendering of the verdict of the trial jury upon the question submitted to it, the court shall either direct a new trial or hearing or make such final order upon the petition as justice requires. Where a committee of the property is appointed, the court shall direct the payment by him out of the funds in his hands of the necessary disbursements of the petitioner, and of such a sum for his costs and counsel fees as it thinks reasonable. (Idem, §§ 2323–2336.)

Powers of Committee.—The committee either of the person or of the property is subject to the direction and control of the court by which he was appointed, with respect to the execution of his duties; and he may be suspended, removed, or allowed to resign in the discretion of the court. A committee of the property cannot alien, mortgage, or otherwise dispose of real property, except to lease for a term not exceeding five years, without the special direction of the court obtained upon proceedings taken for that purpose. (Idem, § 2339.)

THE INSANITY LAW OF NEW YORK.

§ 2. **Definitions.**—When used in this chapter, the term poor person means a person who is unable to maintain himself and having no one legally liable and able to maintain him; the term indigent person means one who has not sufficient property to support himself while insane, and the members of his family lawfully dependent upon him for support; the term institution means any hospital, asylum, building, buildings, house or retreat, authorized by law to have the care, treatment, or custody of the insane; the term commission means the State Commission in Lunacy; the term patient means an insane person committed to an institution according to the provisions of this chapter.

§ 3. **Appointment, Qualifications, Terms of Office, and Salaries of Commissioners.**—There shall continue to be a State Commission in Lunacy, consisting of three commissioners, all of whom shall be citizens of this State. One of them, who shall be president of the commission, shall be a reputable physician, a graduate of an incorporated medical college, of at least ten years' experience in the actual practice of his profession, who has had five years' actual experience in the treatment of mental and nervous diseases or who has had two years' experience in the care and treatment of the committed insane. One of such commissioners shall be a reputable attorney and counsellor-at-law of the courts of this State of not less than ten years' standing. The third commissioner shall be a reputable citizen. Each commissioner shall receive an annual salary of five thousand dollars and twelve hundred dollars in lieu of his travelling and incidental expenses, payable monthly. The full term of office of a commissioner shall be six years. Where the term of office of a commissioner expires at a time other than the last day of December, the term of office of his successor is abridged so as to expire on the last day of December preceding the time when such term would otherwise expire, and the term of office of each commissioner thereafter appointed shall begin on the first day of January. The commissioners shall be appointed by the governor, by and with the advice and consent of the senate.

§ 4. **Office and Clerical Force of Commission.**—The commission shall be provided by the proper authorities with a suitably furnished office in the State capitol, where it shall hold stated meetings at least once in three months. It may hold other meetings, at such office or elsewhere, as it may deem necessary. It may employ a secretary, a stenographer and such other employés as may be necessary. The salaries and reasonable expenses of the commission and of the necessary clerical assistants shall be paid by the treasurer of the State on the warrant of the comptroller, out of any moneys appropriated for the support of the insane.

§ 5. **Official Seal and Execution of Papers.**—The commission shall have an official seal. Every process, order or other paper issued or executed by the commission, may, by the direction of the commission, be attested, under its seal, by its secretary or by any member of the commission, and when so attested shall be deemed to be duly executed by the commission.

§ 6. **General Powers.**—The commission is charged with the execution of the laws relating to the custody, care and treatment of the insane, as provided in this Act, not including feeble-minded persons and epileptics, as such, and idiots. They shall examine all institutions, public and private, authorized by law to receive and care for the insane, and inquire into their methods of government and the management of all such persons therein. They shall examine into the condition of all buildings, grounds and other property connected with any such institution, and into all matters relating to its management. For such purpose each commissioner shall have free access to the grounds, buildings and all books and papers relating to any such institution. All persons connected with any such institution shall give such information, and afford such facilities for any such examination or inquiry as the commissioners may require. The commission may, by order, appoint a competent person to examine the books, papers and accounts, and also into the general condition and management of any institution to the extent deemed necessary and specified in the order. The commission may endeavor to secure legislation from congress to provide more effectually for the removal of alien and non-resident insane, and may expend a reasonable sum therefor, from the moneys appropriated for the use of the hospitals.

§ 6-a. **General Powers as to State Hospitals.**—The commission shall:

1. Have the general management, direction and control of the State hospitals, and of all the property and concerns thereof, and shall see that their design is carried into effect according to law and the rules and regulations adopted therefor.

2. Establish and modify rules and regulations regulating the appointment and duties of officers and employés of each of the State hospitals and the internal discipline and management thereof; but until new rules and regulations are established, the by-laws, rules, and regulations established by the boards of managers shall continue in force. Such rules and regulations shall not conflict with the rules of the State civil service commission in relation to officers and employés of the State hospitals. Such rules and regulations, or any modification thereof, shall take effect when approved by the governor.

3. Maintain an effective supervision and inspection of each of the State hospitals.

4. Acquire and hold in the name of and for the people of the State of New York, by grant, gift, devise or bequest, property to be applied to the maintenance of insane persons in and for the general use of a hospital. Any grant, gift, devise or bequest heretofore

made for such purpose to the managers of a State hospital shall be administered by the State Commission in Lunacy, in accordance with the terms thereof.

5. Transfer, if it deems advisable and subject to the approval of the governor, any of the powers and duties of the superintendent to another officer to be appointed by it. Such officer shall be deemed a resident officer of the hospital for which he is appointed.

§ 7. **Official Visits.**—The commission, or a majority thereof, shall visit every such State hospital jointly or by a majority of the commission and every such private institution by one member of the commission at least twice in each calendar year. Such visits shall be made on such days and at such hours of the day or night, and for such length of time, as the visiting commissioner may choose. But each commissioner may make such other visits as he or the commission may deem necessary. Each visit shall include, to the fullest extent deemed necessary, an inspection of every part of each institution, and all the out-houses, places, buildings, and grounds belonging thereto or used in connection therewith. The commissioners shall, from time to time, make an examination of all the records and methods of administration, the general and special dietary, the stores and methods of supply, and, as far as circumstances may permit, of every patient confined therein, especially those admitted since the preceding visit, giving such as may require it suitable opportunity to converse with the commissioners apart from the officers and attendants. They shall, as far as they deem necessary, examine the officers, attendants and other employés, and make such inquiries as will determine their fitness for their respective duties. At the next regular or special meeting of the commission, after any such visit, the visiting commissioners shall report the result thereof, with such recommendations for the better management or improvement of any such institution, as they may deem necessary. But such recommendations shall not be contrary to the doctrines of the particular school of medicine adopted by such institutions. The commissioners shall, from time to time, meet the managers or responsible authorities of such institutions, or as many of the number as practicable, in conference, and consider, in detail, all questions of management and improvement of the institution, and shall also send to them, in writing, if approved by a majority of the commissioners, such recommendation in regard to the management and improvement of the institution as they may deem necessary or desirable.

§ 8. **Regulations and Forms.**—The commission shall make such regulations in regard to the correspondence of the insane in custody as in its judgment will promote their interests, and it shall be the duty of the proper authorities of each institution to comply with and enforce such rules and regulations. All such insane shall be allowed to correspond without restriction with the county judge and district attorney of the county from which they were committed. The books of record and blank forms for the official use of the hospitals shall be uniform, and shall be approved by the commission.

§ 9. **Annual Report.** The commission shall, annually, report to the legislature its acts and proceedings for the year ending September thirtieth last preceding, with such facts in regard to the management of the institutions for the insane as it may deem necessary for the information of the legislature, including estimates of the amounts required for the use of the State hospitals and the reasons therefor; and also the annual reports made to the commission by the superintendent of each State hospital and by the State Charities Aid Association. The commission shall determine from time to time the capacity of each of the State hospitals and shall incorporate a statement of such capacity in its annual report to the legislature.

§ 10. **State Hospital Districts; how Defined.** The State Commission in Lunacy shall divide the State into as many State hospital districts as there are State hospitals. No county shall be divided in such classification, unless more than one of the existing State hospitals be situated within such county. Whenever the commission shall deem it necessary to more conveniently care for the insane in the various hospitals, it may change the limits of such hospital districts. When a new State hospital shall be established they shall again divide the State into hospital districts. Such hospital districts shall be so defined that the number of patients in each district shall be in proportion, as nearly as practicable, to the accommodations which are or may be provided by the State hospital or hospitals within such district. The hospital districts in which the Long Island State Hospital and the Manhattan State Hospital are located may be subdivided to provide for the commitment of patients to each of the parts of such hospitals, established by section thirty-six of this chapter.

§ 11. **Change of Hospital Districts and Reassignment of Patients.**—When a change or re-establishment of State hospital districts shall be made, or a new State hospital district created, the commission shall make a report thereof, designating the counties included within each district affected thereby, and file the same with the secretary of State, and send a copy to the superintendent of each State hospital, and to each judge of a court of record, each county superintendent of the poor, and each county clerk in the State, to be filed in his office.

§ 12. **Record of Medical Examiners.**—Any physician who receives a certificate as a medical examiner in lunacy shall file such original certificate in the office of the clerk of the county where he resides, and forward a certified copy thereof to the office of the commission within ten days after such certificate is granted. The commission shall keep in its office a record showing the name, residence and certificate of each duly qualified

medical examiner, and shall immediately file in its office, when received, each duly certified copy of a medical examiner's certificate, and advise the examiner of its receipt and filing. No examiner shall be qualified until he has received from the commission an acknowledgment of the receipt and filing of his certificate.

§ 13. **Record of Patients.** —The commission shall keep in its office, and accessible only to the commissioners, their secretary and clerks, except by the consent of the commission or one of its members, or an order of a judge of a court of record, a record showing:

1. The name, residence, sex, age, nativity, occupation, civil condition and date of commitment of every patient in custody in the several institutions for the care and treatment of insane persons in the State, and the name and residence of the person making the petition for commitment, and of the persons signing such medical certificate, and of the judge making the order of commitment.

2. The name of the institution where each patient is confined, the date of admission, and whether brought from home or another institution and if from another institution, the name of such institution, by whom brought, and the patient's condition.

3. The date of the discharge of each patient from such institution since the fifteenth day of May, eighteen hundred and eighty-nine, and whether recovered, improved or unimproved, and to whose care committed.

4. If transferred, for what cause, and to what institution; and if dead, the date and cause of death.

§ 14. **Institutions to Furnish Information to Commission.** —The authorities of the several institutions for the insane shall furnish to the commission the facts mentioned in the last preceding section, and such other obtainable facts relating thereto as the commission may, from time to time, in the just and reasonable discharge of its duties, require of them, with the opinion of the superintendent thereon, if requested. The superintendent or person in charge of such institutions, whether public or private, must, within ten days after the admission of an insane person thereto, cause a true copy of the medical certificate and order on which such person shall have been received, to be made and forwarded to the office of the commission; and when a patient shall be discharged, transferred or shall die therein, such superintendent or person in charge shall, within three days thereafter, send the information to the office of the commission, in accordance with the forms prescribed by it.

§ 15. **Commission to Provide for the Prospective Wants of the Insane.** —The commission shall provide sufficient accommodations for the prospective wants of the poor and indigent insane of the State. To prevent overcrowding in the State hospitals it shall recommend to the legislature the establishment of other State hospitals, in such parts of the State as in its judgment will best meet the requirements of such insane. It shall also furnish to the legislature in each year an estimate of the probable number of patients who will become inmates of the respective State hospitals during the year beginning October first next ensuing, and the cost of all the additional buildings and equipments, if any, which will be required to carry out the provisions of this chapter relating to the care, custody and treatment of the poor and indigent insane of the State. No money shall be expended for the erection of additional buildings, or for unusual repairs or improvements of State hospitals, except upon plans and specifications to be approved by the commission and the governor. The cost of such buildings as are to be occupied by patients erected on the grounds of existing State hospitals, including the necessary equipment for heating, lighting, ventilating, fixtures and furniture, shall in no case exceed the proportion of four hundred and fifty dollars per capita for the patients to be accommodated therein. No municipality of the State shall have the power to modify or change plans or specifications for the erection, repair or improvement of State hospital buildings or the plumbing or sewerage connected therewith. The commission may secure a blanket policy of insurance covering any or all of the buildings, property, or fixtures of the State hospitals.

§ 16. **Director of the Pathologic Institute.** —The commission shall, after a special civil service examination therefor, appoint a director of the pathologic institute, who shall perform, under the direction of the commission, such duties relating to pathologic research as may be required for all the State hospitals for the insane. His office and laboratory shall be in the city of New York. He shall receive an annual salary to be fixed by the commission, subject to the approval of the governor.

§ 17. **Hospital Attorneys.** —The commission shall appoint an attorney for each State hospital, who shall conduct all of the legal business required to be done for or on account of such hospital, at a stated sum to be fixed by the commission, and which shall be a charge upon the maintenance account, and paid in the same manner as other expenses of the hospitals.

INSTITUTIONS FOR THE CARE, TREATMENT AND CUSTODY OF THE INSANE.

SECTION 30. **State Hospitals for the Poor and Indigent Insane.** —There shall continue to be the following hospitals for the care and treatment of the poor and indigent insane of the State which are hereby declared to be corporations; but other insane persons, who are residents of the State, may be admitted when there is room therein for them.[1]

[1] Titles of hospitals are given above, under head NEW YORK.

§ 31. **Abolition of Boards of Managers; Creation of Boards of Visitation.**—The boards of managers of the several State hospitals shall be abolished on the first day of April, nineteen hundred and two, and their powers and duties conferred upon the State Commission in Lunacy, unless otherwise provided by law. There shall be a board of visitation for each State hospital, to consist of five members, to be appointed by the governor. All of such members shall reside within the hospital district of the hospital for which they are appointed. The members of each board of visitation first appointed hereunder shall be appointed for such terms that the term of office of one member shall expire each year, and annually thereafter one member shall be appointed for a full term of five years. If a vacancy shall occur otherwise than by expiration of term, the appointment of a member to fill such vacancy shall be for the unexpired term of his predecessor. No person shall be eligible as a member of any such board who is an elective State officer, or a member of the legislature, and if any such member shall become such elective State officer, or a member of the legislature, his office as a member of such board shall become vacant. A member of a board of visitation may be removed at the pleasure of the governor. The members of the boards of visitation first appointed hereunder shall be appointed by the governor on or before April fifteenth, nineteen hundred and two.

§ 32. **Powers and Duties of Boards of Visitation.**—Each of such boards of visitation shall elect from its members a president and a secretary. The president shall preside at all meetings, and the secretary shall enter the minutes of such meetings in a book to be provided for that purpose. Each of such boards shall, by a majority of its members, visit and inspect each hospital for which it is appointed at least monthly, and as a board, or by any of its members, when directed by the governor. Such board shall make a written report in duplicate to the governor and to the Commission in Lunacy within ten days after each visitation, to be signed by each member making such visitation. Such report shall state in detail the condition of the hospital visited and of its inmates, and such other matters pertaining to the management and affairs thereof as in the opinion of the board should be brought to the attention of the governor, or the Commission in Lunacy, and may contain recommendations as to needed improvements in the hospital or its management. The resident officers shall admit such board, or the members thereof, into every part of the hospital and its buildings, and exhibit to them, upon demand, all the property, supplies, books, papers, accounts, and writings belonging to the hospital, or pertaining to its business, management, discipline, or government, and furnish copies, abstracts, and reports whenever required by them.

§ 33. **Expenses of Boards of Visitation.**—The members of a board of visitation shall not receive any compensation for their services, but shall receive their actual necessary travelling and other expenses, to be paid by the State treasurer, on the warrant of the comptroller, out of any moneys appropriated therefor.

§ 34. **Officers.**—The Commission in Lunacy, pursuant to the civil service law and the rules and regulations of the State Civil Service Commission, shall appoint for each hospital, as often as vacancies occur therein, a superintendent and a steward. The superintendent shall be a well educated physician and a graduate of an incorporated medical college, of at least five years' actual experience in an institution for the care and treatment of the insane. The superintendents and all assistant physicians of homeopathic hospitals for the insane shall be homeopathic physicians, but such homeopathic physicians shall not be eligible to appointment in or transfer to State hospitals that are not for homeopathic treatment. Each superintendent shall be the treasurer of the State hospital for which he is appointed, and before entering upon his duties as such treasurer shall file with the comptroller of the State his undertaking to the people in an amount and with sureties to be approved by the State comptroller, to the effect that he will faithfully perform his trust as such treasurer. The superintendent or steward may be removed by a vote of a majority of the commission for cause stated in writing, and after an opportunity has been given him to be heard, and such action shall be final. Such commission may issue subpœnas and take and hear testimony in respect to charges made against either of such officers. A witness attending before such commission shall be entitled to the same fees as a witness attending before a court of record, or a judge thereof, which shall be paid as other hospital charges. Except as otherwise provided in this section, superintendents and assistant physicians may, with the approval of the governor, be transferred by the commission from one State hospital to another. On the first day of April, nineteen hundred and two, the office of treasurer in each of the State hospitals shall be abolished, and their powers and duties shall be conferred upon the superintendent. A superintendent or a steward in office on such date shall be continued in office until removed pursuant to law, notwithstanding the change hereby made in the manner of his appointment.

§ 35. **General Powers and Duties of Superintendent.**—The superintendent of each hospital shall be its chief executive officer, and in his absence or sickness the first assistant physician or other officer designated by the superintendent shall perform the duties and be subject to the responsibilities of the superintendent. Subject to the by-laws and regulations established by the commission the superintendent shall have the general superintendence of the buildings, grounds, and farm, together with their furniture, fixtures, and stock, and the direction and control of all persons therein, and subject to such by-laws and regulations shall:

1. Personally maintain an effective supervision and inspection of all parts of the hos-

pital and generally direct the care and treatment of the patients. To this end the superintendent shall personally examine the condition of each patient, within five days after his admission to the hospital, and shall regularly visit all of the wards or apartments for patients at such times as the rules and regulations of the hospital shall prescribe.

2. Appoint such resident officers, except the steward, including a woman physician, and such employés as he may think proper and necessary for the economical and efficient performance of the business of the hospital and prescribe their duties and, for cause stated in writing, after an opportunity to be heard, discharge any of such employés in his discretion. The number of such resident officers and employés shall be determined by the commission. The commission may, with the approval of the governor, abolish the office of any such resident officers or employés. The superintendent may, with the approval of the commission, remove any resident officer, except the steward, for cause stated in writing, after an opportunity to be heard, and such action shall be final. Upon any such removal he shall make a record thereof, with the reasons therefor, under the appropriate head in one of the books of the hospital. The superintendent, assistant physicians, including the woman physician, steward and matron shall constantly reside in the hospital, or on the premises, and shall be designated the resident officers of the hospital. The assistant physicians, including the woman physician, shall be graduates of an incorporated medical college, and shall possess such other qualifications as may be required by law.

3. Transmit, by mail, to the commission in lunacy, within five days after any such discharge, information of such discharge, and of the cause thereof. The commission shall preserve the name of such officer, or employé, with the facts relating to his discharge, in a book provided for that purpose.

4. Designate hospital attendants or employés to act as special policemen, whose duty it shall be, under the orders of the superintendent, to arrest and return to the hospital insane persons who may escape therefrom, and to preserve peace and good order in such hospital and to fully protect the grounds, buildings, and patients. Such attendants and employés, acting as policemen, shall possess all the powers of peace officers on the grounds and premises of such hospitals and to the extent of one hundred yards beyond such grounds. The designation of such attendants and employés as special policemen, in pursuance hereof, shall not be deemed to supersede, on the grounds and premises of such hospital, the authority of peace officers of the jurisdiction within which such hospital is located.

5. Give such orders and instructions as he may deem best calculated to insure good conduct, fidelity, and economy in every department of labor and expense.

6 Maintain salutary discipline among all who are employed in the institution and enforce strict compliance with his instructions and uniform obedience to all rules and regulations of the hospital.

7. Establish and supervise a training school for attendants and nurses, under rules and regulations of the hospital.

8. Cause full and fair accounts and records of all his doings and of the entire business and operations of the hospital, to be kept regularly, from day to day, in books provided for that purpose.

9. See that all such accounts and records are fully made up to the last day of September in each year, and that the principal facts and results, with his report thereon, be presented to the commission within thirty days thereafter. The commission may prescribe the form of and the subjects to be embraced in such reports. Such superintendent shall make other reports at such times, in such manner, and in respect to such matters as the commission may direct.

10. Keep a book, in which he shall cause to be entered at the time of reception of any patient, his name, residence, and occupation, and the date of such reception, by whom brought and by what authority and on whose petition committed, and an abstract of all orders, warrants, requests, petitions, certificates, and other papers accompanying such person.

§ 36. **Special Provisions Relating to Long Island State Hospital and Manhattan State Hospital.**—The Long Island State Hospital is divided into two parts: The part located at Kings Park shall be known as "Long Island State Hospital at Kings Park"; the part located at Flatbush in the borough of Brooklyn, city of New York, shall be known as "Long Island State Hospital at Flatbush." Manhattan State Hospital is divided into three parts: The part located on Ward's Island known as the men's department shall be known as "Manhattan State Hospital East"; the part located on Ward's Island known as the women's department shall be known as "Manhattan State Hospital West"; the part located at Central Islip as "Manhattan State Hospital at Central Islip." After the tenth day of April, nineteen hundred and two, there shall be one superintendent and one steward of the two parts of the Manhattan State Hospital located on Ward's Island, whose powers and duties, subject to the provisions of this chapter, shall extend over both parts of such hospital located upon said island. The commission shall designate one of the superintendents and one of the stewards of one of such parts, in office on April first, nineteen hundred and two, to act as superintendent and steward for both such parts, and upon such designation the terms of office of the superintendent and steward of the other part of such hospital upon said island, shall cease and determine. Each part of each of such

hospitals shall, except as otherwise provided in this section, be deemed a State hospital, and all the provisions of this chapter relating to the management, maintenance, and control of State hospitals and the appointment of resident officers, attendants, and employés therein shall apply to each such part, except that but one board of visitation shall be appointed for the Manhattan State Hospital, and but one board of visitation for the Long Island State Hospital. Patients shall be committed to and received at each part of Long Island State Hospital and Manhattan State Hospital in accordance with rules to be established by the State Commission in Lunacy. The commission may also adopt rules regulating the transfer of such patients from one part to another of such hospitals.

§ 37. **Meeting of Superintendents.** The superintendents or other officers of the several State hospitals, or any of them, shall meet, upon the call of the commission, at the office of the commission at Albany, or at such other place as may be designated by it, to consult with such commission with reference to matters relating to the care and maintenance of the State hospitals, and particularly with reference to the purchase of supplies for their use.

§ 38. **Salaries of Officers and Wages of Employés.**—The commission, from time to time, with the approval in writing of the governor, secretary of State, and comptroller, shall fix the annual salaries of the resident officers of the State hospitals, which shall be uniform for like service. They shall classify the other officers and employés into grades, and determine the salaries and wages to be paid in each grade, which shall be uniform in all the hospitals. The salaries and wages shall be included in the estimates and paid in the same manner as other expenses of the State hospitals. Food supplies shall be allowed to officers and employés and the families of the superintendents, first assistant physicians, and stewards. Food supplies shall continue to be allowed the families of the assistant physicians residing at the hospitals on January first, eighteen hundred and ninety-six. Such families shall consist only of the wives and minor children of such officers; no other persons, except those regularly employed, shall be allowed rooms and maintenance, except at a rate to be fixed by the commission; such supplies shall be drawn from the supplies provided for general hospital use. With the approval of the commission, officers or employés of State hospitals may be permitted to live outside of such hospitals, and shall receive such sums in lieu of the quarters or supplies furnished by the hospitals, as may be equitable.

§ 39. **Monthly Estimate of Expenses: Contingent Fund.**—The superintendent of each of the State hospitals shall at least once in each two months as the commission may determine cause to be prepared triplicate estimates in minute detail, of the expenses required for the hospital of which he is the superintendent, for the ensuing two months. He shall submit two of such triplicates to the commission and file the third copy in the office of the superintendent. The commission may revise estimates for supplies or other expenditures either as to quantity, quality, or the estimated cost thereof, and shall certify that it has carefully examined the same and that the articles contained in such estimate, as approved or revised by it, are actually required for the use of the hospital, and shall thereupon present such estimate and certificate to the comptroller. Upon the revision and approval of such estimate by the commission, the comptroller shall authorize the superintendent as treasurer to make drafts on the comptroller, as the money may be required for the purposes mentioned in such estimates, which drafts shall be paid on the warrant of the comptroller out of the funds in the treasury of the State held for the care of the insane and the maintenance of State hospitals. In every such estimate, there shall be a sum named, not to exceed one thousand dollars, as an emergency fund for which no minute detailed statement need be made. No money shall be expended for the use of any of the State hospitals, except as provided in this section. Libraries may be furnished to any State hospital by the regents of the University of the State of New York, subject to regulations adopted by them and the commission, the expense of which shall be included in the bi-monthly estimates of the hospital. Any general expense necessarily incurred by the commission for or on account of the State hospitals shall be apportioned to such hospitals on the basis of the number of patients, and included in the estimates of such hospitals made as provided in this section under the direction of the commission.

§ 40. **Powers and Duties of Superintendent as Treasurer.**—The superintendent as treasurer of each hospital shall, subject to the rules and regulations of the commission:

1. Have the custody of all moneys received from the comptroller on account of estimates made by the superintendent and revised and approved by the commission, and keep an accurate account thereof.

2. Have the custody of all bonds, notes, mortgages, and other securities and obligations belonging to the hospital.

3. Receive all money for the care and treatment of private patients and other sources of revenue of the hospital.

4. Deposit all such money in a bank designated by the comptroller conveniently near the hospital, in his name as treasurer, and send each month to the comptroller and to the commission a statement showing the amount so received and deposited and from whom and for what received, and when such deposits were made. Such statement of deposit shall be certified by the proper officer of the bank receiving such deposit. The superintendent as treasurer shall make an affidavit to the effect that the sum so deposited is all the money received by him, from any source of hospital income up to the time of the last

deposit appearing on such statement. A bank designated by the comptroller to receive such deposits shall, before any deposit is made, execute a bond to the people of the State, in a sum approved by the comptroller, for the safe keeping of the funds deposited.

5. Pay out the money deposited for the uses of the State hospital upon the voucher of the steward.

6. Keep full and accurate accounts of all receipts and payments, in the manner directed in the by-laws and according to books and forms prescribed and furnished by the commission.

7. Balance all accounts on his books, annually, on the last day of September, and make a statement thereof and an abstract of the receipts and payments of the past year and deliver the same, within ten days, to the commission.

8. Render an account of the state of the books and the funds and other property in his custody whenever required by the commission.

9. Execute a release and satisfaction of a mortgage, judgment, or other lien or debt in favor of the hospital, when paid.

10. Receive all moneys for or on account of the sale of lands of the hospital of which he is treasurer.

§ 41. **Monthly Statements of Receipts and Expenditures; Vouchers.**—The superintendent, as treasurer of each State hospital, shall, on or before the fifteenth day of each month, make to the comptroller and to the commission a full and perfect statement of all the receipts and expenditures, specifying the several items, for the last preceding calendar month. Such statement shall be verified by the affidavit of the treasurer attached thereto, in the following form:

I, ... , treasurer of the . . . . State hospital, do solemnly swear that I have deposited in the bank designated by law for such purpose, all the moneys received by me on account of the hospital during the last month, and I do further swear that the foregoing is a true abstract of all the moneys received and payments made by me or under my direction as such treasurer during the month ending on the . . . . day of . . . . , 18 . .

There shall also be attached thereto the affidavit of the steward, to the effect that the goods and other articles therein specified were purchased and received by him, or under his directions, at the hospital; that the goods were purchased at a fair cash market price and paid for in cash, or on credit, not exceeding thirty days, and that he, or any person in his behalf, had no pecuniary or other interest in the articles purchased; that he received no pecuniary or other benefit therefrom in the way of commission, percentage, deductions, or presents, or in any other manner whatever, directly or indirectly; that the articles contained in such bill were received at the hospital; that they conformed in all respects to the invoiced goods received and ordered by him, both in quality and quantity. Such statement shall be accompanied by the vouchers showing the payment of the several items contained in the statement and the approval thereof by the superintendent, the amount of such payment and for what the payment was made. Such approval may be contained on an audit sheet, which shall refer to each voucher approved by the superintendent, giving the number of voucher, the name of the claimant, and the amount at which it was approved. Such vouchers shall be examined by the commission and compared with the estimates made for the month for which the statement is rendered, and if found correct shall be indorsed and forwarded by the commission, with the statement to the comptroller. If any voucher is found objectionable, the comptroller shall indorse his disapproval thereon, with the reason therefor, and return it to the treasurer, who shall present it to the superintendent for correction, and when corrected return it to the comptroller. All such vouchers shall be filed in the office of the comptroller.

§ 42. **Actions to Recover Moneys Due the Hospital.**—The superintendent as treasurer of any State hospital may bring an action in the name of the hospital, to recover for the use thereof:

1. The amount due upon any note or bond in his hands belonging to the hospital.

2. The amount charged and due, according to the by-laws of the hospital, for the support of any patient therein, or for actual disbursements made in his behalf for necessary clothing and travelling expenses, and to enforce any liability created by statute for the care and support of the insane.

§ 43. **General Powers and Duties of the Steward.**—The steward, under the direction of the superintendent, and subject to the rules and regulations of the commission, shall be accountable for the careful keeping and economical use of all furniture, stores, and other articles provided for the hospital, and under the direction of the superintendent, and subject to such rules and regulations, shall:

1. Make all purchases for the hospital and preserve the original bills and receipts thereof, and keep full and accurate accounts of the same.

2. Prepare and keep the pay-rolls of the hospital.

3. Keep the accounts for the support of patients and expenses incurred in their behalf, and furnish the treasurer statements thereof as they fall due.

4. Notify the treasurer of the death or discharge of any reimbursing or pay patient, within five days after such death or discharge.

§ 44. **Purchases and Contracts.**—All purchases of supplies for the use of the hospital shall be made for cash or on credit or time, not exceeding sixty days; every voucher shall

be duly filled up, and with every abstract of vouchers paid there shall be proof on oath that the voucher was properly filled up and the money paid. No expenditure for supplies or other purposes shall be made for the benefit of such hospitals, by contract or otherwise, unless in conformity with the provisions of this Act in relation to estimates. No member of the commission or officer of a hospital shall be interested, directly or indirectly, in the furnishing of material, labor, or supplies for the use of the hospital, nor shall any such officer act as attorney or counsel for such hospital. Contracts subject to the approval of the commission shall be entered into jointly, by the stewards of the State hospitals, for such staple articles of supplies as it may be found feasible by the commission to purchase for the use of the hospitals. Such contracts shall not be let except in conformity with the provisions of this Act relating to estimates. The State hospitals may manufacture such supplies and materials to be used in any of such hospitals as can be economically made therein. All goods for the use of the hospitals shall be bought, as far as practicable, of manufacturers or their immediate agents. All contracts, if let, shall, subject to the provisions of section thirty-nine, relating to estimates, be awarded to the lowest responsible bidders. A member of the commission or an officer, or employe of a State hospital shall not receive a gift or reward for himself or the hospital from any person, firm, or corporation dealing in goods or supplies suitable or necessary for the use of the hospital. All purchases and contracts made and executed in pursuance of law, prior to April first, nineteen hundred and two, shall thereafter be given full force and effect, notwithstanding the change in the management of the State hospitals.

§ 45. **Official Oath.** Each superintendent and steward of a hospital, before entering upon his duties as such, shall take the constitutional oath of office and file the same in the office of the secretary of State.

§ 46. **Actions Against Commissioners in Lunacy, or Officers of State Hospitals.**—No civil action shall be brought in any court against the commission or a commissioner in lunacy, or an officer of a State hospital, for alleged damages because of any act done or failure to perform any Act, while discharging their official duties, without leave of a judge of the Supreme Court, first had and obtained. Any just claim for damages against such commission or commissioner, officer, or employé for which the State would be legally or equitably liable, shall be paid out of any moneys appropriated for the care of the insane.

§ 47. **Private Institutions for the Insane.** No person, association, or corporation shall establish or keep an institution for the care, custody, or treatment of the insane, for compensation or hire, without first obtaining a license therefor from the commission. Every application for such license shall be accompanied by a plan of the premises proposed to be occupied, describing the capacities of the buildings for the uses intended, the extent and location of grounds appurtenant thereto, and the number of patients proposed to be received therein, with such other information, and in such form, as the commission may require. The commission shall not grant any such license without first having made an examination of the premises proposed to be licensed, and being satisfied that they are substantially as described, and are otherwise fit and suitable for the purposes for which they are designed to be used, and that such license should be granted. The commission may, at any and all times, examine and ascertain how far a licensed institution is conducted in compliance with the license therefor, and after due notice to the institution and opportunity for it to be heard, the commission having made a record of the proceeding upon such hearing, may, if the interests of the inmates of the institution so demand, for just and reasonable cause then appearing and to be stated in its order, amend or revoke any such license by an order to take effect within such time after the service thereof upon the licensee, as the commission shall determine.

§ 48. **Recommendations of Commission.**—The authorities of each institution for the insane shall place on file in the office of the institution the recommendations made by the commissioners as a result of their visit, for the purpose of consultation by such authorities, and for reference by the commissioners upon their visits.

§ 49. **Visitors to State Hospitals.**—Justices of the Supreme Court are authorized to appoint visitors to State hospitals, upon nomination of the State Charities Aid Association, as provided by law.

* * * * * * *

§ 52. **Acquisition of Real Property for Use of State Hospitals by Condemnation.**—The State Commission in Lunacy may acquire, under the condemnation law, such real estate, right or interest therein as may be necessary for the construction, maintenance and accommodation of a State hospital, if unable to agree with the owner thereof for its purchase. The proceedings for the purpose of acquiring such real estate, right or interest therein shall be instituted and maintained in the name of the People of the State of New York, by the attorney-general or by such counsel as the governor or attorney-general may designate for that purpose, upon the certificate of such commission as to the necessity of acquiring such real estate, right or interest therein, approved and endorsed by the governor.

§ 53. **Erection, Repairs, and Improvements of State Hospital Buildings.**—All plans and specifications for the erection, repairs, and improvements of State hospital buildings shall be prepared by the State architect and he may employ such experts, engineers, and assistants as may be necessary for the proper conduct of such work, whose compensation shall be fixed by said architect, with the approval of the commission, and shall be paid by the

treasurer of the State hospital where the work is to be performed, out of any moneys allotted by the commission for that purpose. Contracts for such erection, repairs, and improvements may be let by the commission, subject to the approval of the governor and comptroller, for the whole or any part of the work to be performed, and in the discretion of the commission such contracts may be sublet. The comptroller and the commission shall determine to what extent and for what length of time advertisements are to be inserted in newspapers for proposals for the erection, repairs, or improvements of State hospital buildings. A preliminary deposit, or certified check drawn upon some legally incorporated bank in this State, shall in all cases be required as an evidence of good faith, upon all proposals for buildings, repairs, and improvements, to be deposited with the treasurer of the hospital for which the work is to be performed, in an amount to be determined by the State architect. All contracts for the erection, repairs, or improvements to hospitals shall contain a clause that the contract shall only be deemed executory to the extent of the moneys available, and no liability shall be incurred by the State beyond the moneys available for the purpose. The commission is directed, to the fullest extent deemed practicable, to provide additional buildings for the removal of the insane from the Flatbush department of the Long Island State Hospital.

§ 54. **Streets and Railroads Through Hospital Lands.** No public street or road for railroad or other purposes shall be opened through the lands of a State hospital unless the legislature by special law consents thereto.

ARTICLE III.

COMMITMENT, CUSTODY AND DISCHARGE OF THE INSANE.

§ 60. **Order for Commitment of an Insane Person.**—A person alleged to be insane, and who is not in confinement on a criminal charge, may be committed to and confined in an institution for the custody and treatment of the insane, upon an order made by a judge of a court of record of the city or county, or a justice of the Supreme Court of the judicial district, in which the alleged insane person resides or may be, adjudging such person to be insane, upon a certificate of lunacy, made by two qualified medical examiners in lunacy, accompanied by a verified petition therefor, or upon such certificate and petition, and after a hearing to determine such question, as provided in this article. The commission shall prescribe and furnish blanks for such certificate and petitions, which shall be made only upon such blanks. An insane person shall be committed only to a State hospital a duly licensed institution for the insane, or the Matteawan State Hospital, or to the care and custody of a relative or committee, as hereinafter provided. No idiot shall be admitted to or confined in a State hospital. But any epileptic or feeble-minded person becoming insane may be committed as an insane person to a State hospital for custody and treatment.

§ 61. **Medical Examiners in Lunacy; Certificates of Lunacy.**—The certificate of lunacy must show that such person is insane and must be made by two reputable physicians, graduates of an incorporated medical college, who have been in the actual practice of their profession at least three years, and have filed with the commission a certified copy of the certificate of a judge of a court of record, showing such qualifications in accordance with forms prescribed by the commission.

Such physicians shall jointly make a final examination of the person alleged to be insane within ten days next before the granting of the order. The date of the certificate of lunacy shall be the date of such joint examination. Such certificate of lunacy shall be in the form prescribed by the commission, and shall contain the facts and circumstances upon which the judgment of the physicians is based and show that the condition of the person examined is such as to require care and treatment in an institution for the care, custody, and treatment of the insane.

Neither of such physicians shall be a relative of the person applying for the order of the person alleged to be insane, or a manager, superintendent, proprietor, officer, stockholder, or have any pecuniary interest, directly or indirectly, or be an attending physician in the institution to which it is proposed to commit such person.

§ 62. **Proceedings to Determine the Question of Insanity.**—Any person with whom an alleged insane person may reside or at whose house he may be, or the father or mother, husband or wife, brother or sister, or the child of any such person, and any overseer of the poor of the town, and superintendent of the poor of the county in which any such person may be, may apply for such order, by presenting a verified petition containing a statement of the facts upon which the allegation of insanity is based, and because of which the application for the order is made. Such petition shall be accompanied by the certificate of lunacy of the medical examiners, as prescribed in the preceding section. Notice of such application shall be served personally, at least one day before making such application, upon the person alleged to be insane, and if made by an overseer or superintendent of the poor, also upon the husband or wife, father or mother or next of kin of such alleged insane person, if there be any such known to be residing within the county, and if not, upon the person with whom such alleged insane person may reside, or at whose house he may be. The judge to whom the application is to be made may dispense with such personal service, or may direct substituted service to be made upon some person to be designated by him. He shall state in a certificate to be attached to the petition his

reason for dispensing with personal service of such notice, and if substituted service is directed, the name of the person to be served therewith.

The judge to whom such application is made may, if no demand is made for a hearing in behalf of the alleged insane person, proceed forthwith to determine the question of insanity, and if satisfied that the alleged insane person is insane, may immediately issue an order for the commitment of such person to an institution for the custody and treatment of the insane. If, however, it appears that such insane person is harmless and his relatives or a committee of his person are willing and able to properly care for him at some place other than such institution, upon their written consent, the judge may order that he be placed in the care and custody of such relatives or such committee. Such judge may, in his discretion, require other proofs in addition to the petition and certificate of the medical examiners.

Upon the demand of any relative or near friend in behalf of such alleged insane person, the judge shall, or he may upon his own motion, issue an order directing the hearing of such application before him at a time not more than five days from the date of such order, which shall be served upon the parties interested in the application and upon such other persons as the judge, in his discretion, may name. Upon such day, or upon such other day to which the proceeding shall be regularly adjourned, he shall hear the testimony introduced by the parties and examine the alleged insane person if deemed advisable, in or out of court, and render a decision in writing as to such person's insanity. If it be determined that such person is insane, the judge shall forthwith issue his order committing him to an institution for the custody and treatment of the insane, or make such other order as is provided in this section. If such judge can not hear the application, he may, in his order directing the hearing, name some referee, who shall hear the testimony and report the same forthwith, with his opinion thereon, to such judge, who shall, if satisfied with such report, render his decision accordingly. If the commitment be made to a State hospital, the order shall be accompanied by a written statement of the judge as to the financial condition of the insane person and of the persons legally liable for his maintenance as far as can be ascertained. The superintendent of such State hospital shall be immediately notified of such commitment, and he shall, at once, make provisions for the transfer of such insane person to such hospital.

The petition of the applicant, the certificate in lunacy of the medical examiners, the order directing a further hearing as provided in this section, if one be issued, and the decision of the judge or referee, and the order of commitment shall be presented at the time of the commitment to the superintendent or person in charge of the institution to which the insane person is committed, and verbatim copies shall be forwarded by such superintendent or person in charge and filed in the office of the State Commission in Lunacy. The relative, or committee, to whose care and custody any insane person is committed, shall forthwith file the petition, certificate, and order, in the office of the clerk of the county where such order is made, and transmit a certified copy of such papers, to the Commission in Lunacy, and procure and retain another such certified copy.

The superintendent or person in charge of any institution for the care and treatment of the insane may refuse to receive any person upon any such order, if the papers required to be presented shall not comply with the provisions of this section, or if in his judgment such person is not insane within the meaning of this statute, or if received, such person may be discharged by the commission. No person shall be admitted to any such institution under such order after the expiration of five days from and inclusive of the date thereof.

Notwithstanding the requirements of this section that an alleged insane person be duly committed by an order of the court, in a case where the condition of such person is such that it would be for his benefit to receive immediate care and treatment, or if he is dangerously insane so as to render it necessary for public safety that he be immediately confined, he shall be forthwith received by a state institution authorized by law to care for the insane. In such case such insane person shall be so received by such institution upon a certificate of lunacy, executed by two medical examiners in lunacy after the examination and in the manner provided in the preceding section, and upon a petition made by the person authorized by this section to apply to a court for an order of commitment. By virtue of such certificate of lunacy and such petition such insane person may be retained in such institution for a period not to exceed five days. Prior to the expiration of such time an order for his commitment must be obtained in the manner provided by this section. The certificate of lunacy executed by such physicians must contain adequate reasons why the insane person should be immediately received in an institution for the insane for treatment. The superintendent or person in charge of any such institution may refuse to receive such insane person upon such certificate and petition, if in his judgment the reasons stated in the certificate, or the condition of the patient are not sufficient, or is not of such character, as to make it necessary that the patient should receive immediate treatment.

§ 63. **Appeal from Order of Commitment.**—If a person ordered to be committed pursuant to this chapter, or any friend in his behalf, is dissatisfied with the final order of a judge or justice committing him, he may, within ten days after the making of such order, appeal therefrom to a justice of the Supreme Court other than the justice making the order, who shall cause a jury to be summoned as in the case of proceedings for the ap-

pointment of a committee for an insane person, and shall try the question of such insanity in the same manner as in proceedings for the appointment of a committee. Before such appeal shall be heard, such person shall make a deposit or give a bond, to be approved by a justice of the Supreme Court, for the payment of the costs of the appeal, if the order of commitment is sustained. If the verdict of the jury be that such person is insane, the justice shall certify that fact and make an order of commitment as upon the original hearing. Such order shall be presented, at the time of the commitment of such insane person, to the superintendent or person in charge of the institution to which the insane person is committed, and a copy thereof shall be forwarded to the commission by such superintendent or person in charge and filed in the office thereof. Proceedings under the order shall not be stayed pending an appeal therefrom, except upon an order of a justice of the Supreme Court, and made upon a notice, and after a hearing, with provisions made therein for such temporary care or confinement of the alleged insane person as may be deemed necessary.

If a judge shall refuse to grant an application for an order of commitment of an insane person proved to be dangerous to himself or others, if at large, he shall state his reasons for such refusal in writing, and any person aggrieved thereby may appeal therefrom in the same manner and under like conditions as from an order of commitment.

§ 64. **Costs of Commitment.**—The costs necessarily incurred in determining the question of the insanity of a poor or indigent person and in securing his admission into a State hospital, and the expense of providing proper clothing for such person, in accordance with the rules and regulations adopted by the commission, shall be a charge upon the town, city, or county securing the commitment. Such costs shall include the fees allowed by the judge or justice ordering the commitment to the medical examiners. If the person sought to be committed is not a poor or indigent person, the costs of the proceedings to determine his insanity and to secure his commitment, as provided in this article, shall be a charge upon his estate, or shall be paid by the persons legally liable for his maintenance. If in such proceedings the alleged insane person is determined not to be insane, the judge or justice may, in his discretion, charge the costs of the proceedings to the person making the application for an order of commitment, and judgment may be entered for the amount thereof and enforced by execution against such person.

§ 65. **Liability for Care and Support of Poor and Indigent Insane.**—All poor and indigent insane persons not in confinement under criminal proceedings, shall, without unnecessary delay, be transferred to a State hospital and there wholly supported by the State. The costs necessarily incurred in the transfer of patients to State hospitals shall be a charge upon the State. The commission shall secure from the patient's estate and from relatives or friends who are liable or may be willing to assume the costs of support of inmates of State hospitals supported by the State, reimbursement, in whole or in part, of the money thus expended, either directly or through the treasurers of the respective hospitals, as provided in section forty-two of this Act. (Thus amended by chapter 546, Laws of 1901.)

The commission may appoint agents, whose duty it shall be to secure from relatives and friends who are liable therefor, or who may be willing to assume the cost of support of any of the inmates of State hospitals as are being supported by the State, reimbursement in whole or in part of the money so expended. The compensation of each agent shall not exceed five dollars a day, and the necessary travelling and other incidental expenses incurred by him, to be approved by the comptroller. The commission may fix a rate to be paid for the support of the inmates of State hospitals by relatives liable for such support or by those not liable for such support, but willing to assume the cost thereof; but such rate shall be sufficient to cover a proper proportion of the cost of maintenance and of necessary repairs and improvements. The maintenance of any inmate of a State hospital committed thereto upon a court order arising out of any criminal action or proceeding shall be paid by the county from which such inmate was committed.

§ 66. **Liability for the Care and Support of the Insane Other Than the Poor and Indigent.**—The father, mother, husband, wife, and children of an insane person, if of sufficient ability, and the committee or guardian of his person or estate, if his estate is sufficient for the purpose, shall cause him to be properly and suitably cared for and maintained.

The commission and the superintendent of the poor of the county, and the overseer of the poor of the town where any such insane person may be, or in the city of New York, the Commissioners of Public Charities may inquire into the manner in which any such person is cared for and maintained: and if, in the judgment of any of them, he is not properly or suitably cared for, may apply to a judge of a court of record for an order to commit him to a State hospital under the provisions of this article, but such order shall not be made unless the judge finds and certifies in the order that such insane person is not properly or suitably cared for by such relative or committee, or that it is dangerous to the public to allow him to be cared for and maintained by such relative or committee.

The costs and charges of the commitment and transfer of such insane person to a State hospital shall be paid by the committee, or the father, mother, husband, wife, or children of such person, to be recovered in an action brought in the name of the people by the commission, the superintendent of the poor of the county, or the overseer of the poor of the town where such insane person may be, or in the city of New York in the name of the

Commissioners of Public Charities. In all claims of the State upon relatives liable for the support of a patient, or upon moneys or property held by such patient, the State shall be deemed a preferred creditor.

§ 67. **Duties of Local Officers in Regard to Their Insane.**—All county superintendents of the poor, overseers of the poor, and other city, town, or county authorities having duties to perform relating to the insane poor, are charged with the duty of seeing that all poor and indigent insane persons within their respective municipalities are timely granted the necessary relief conferred by this chapter, and when so ordered by a judge, as herein provided, or by the commission, shall see that they are, without unnecessary delay, transferred to the proper institutions provided for their care and treatment as the wards of the State. Before sending a person to any such institution, they shall see that he is in a state of bodily cleanliness and comfortably clothed with new clothing, in accordance with the regulations prescribed by the commission. The commission may, by order, direct that any person it deems unsuitable therefor shall not be so employed or act as such attendant.

Each patient shall be sent to the State hospital within the district embracing the county from which he is committed, except that the commission may, in their discretion, direct otherwise; but private or public insane patients, for whom homeopathic care and treatment may be desired by their relatives, friends, or guardians, may be committed to the Middletown State Homeopathic Hospital, or to the Gowanda State Homeopathic Hospital, from any of the counties of the State, in the discretion of the judge granting the order of commitment; and the hospital to which any patient is ordered to be sent shall, by and under the regulations made by such commission, send a trained attendant to bring the patient to the hospital. Each female committed to any institution for the insane shall be accompanied by a female attendant, unless accompanied by her father, brother, husband, or son. After the patient has been delivered to the proper officers of the hospital, the care and custody of the municipality from which he is sent shall cease.

§ 68. **Duty of Committee and Others to Care for the Insane; Apprehension and Confinement of a Dangerous Insane Person.**—When an insane person is possessed of sufficient property to maintain himself, or his father, mother, husband, wife, or children are of sufficient ability to maintain him, and his insanity is such as to endanger his own person, or the person and property of others, the committee of his person and estate, or such father, mother, husband, wife, or children must provide a suitable place for his confinement, and there maintain him in such manner as shall be approved by the proper legal authority. The county superintendent of the poor and the overseers of the poor of towns and cities, the Commissioners of Public Charities in the city of New York, and the Commissioners of Charities and Correction in the city of Brooklyn, are required to see that the provisions of this section are carried into effect in the most humane and speedy manner.

Upon the refusal or neglect of a committee, guardian, or relative of an insane person to cause him to be confined, as required in this chapter, the officers named in this section shall apply to a judge of a court of record of the city or county, or to a justice of the Supreme Court of the judicial district in which such insane person may reside or be found, who, upon being satisfied, upon proper proofs, that such person is dangerously insane and improperly at large, shall issue a precept to one or more of the officers named, commanding them to apprehend and confine such insane person in some comfortable and safe place; and such officers in apprehending such insane person shall possess all the powers of a peace officer executing a warrant of arrest in a criminal proceeding. Unless an order of commitment has been previously granted, such officers shall forthwith make application for the proper order for his confinement to the proper institution for the care, custody, and treatment of the insane, as authorized by this chapter, and if such order is granted, such officer shall take the necessary legal steps to have him transferred to such institution. In no case shall any such insane person be confined in any other place than a State hospital or duly licensed institution for the insane, for a period longer than ten days, nor shall such person be committed as a disorderly person to any prison, jail, or lockup for criminals, unless he be violent and dangerous, and there is no other suitable place for his confinement; nor shall he be confined in the same room with a person charged with or convicted of crime.

Any person apparently insane, and conducting himself in a manner which in a sane person would be disorderly, may be arrested by any peace officer and confined in some safe and comfortable place until the question of his sanity be determined, as prescribed by this chapter. The officer making such arrest shall immediately notify the superintendent of the poor of the county, or the overseers of the poor of the town or city, or, in the city of New York, the Commissioners of Public Charities, or, in the city of Brooklyn, the Commissioners of Charities and Correction, who shall forthwith take proper measures for the determination of the question of the insanity of such person.

§ 69. **Patients Admitted under Special Agreement.**—The commission may authorize the superintendent of a State hospital to admit thereto, under special agreement, insane patients, who are residents of the State, other than poor and indigent insane persons, when there is room for such insane therein. But no patient shall be permitted to occupy more than one room in any State hospital. Such patients, when so received, shall be subject to the general rules and regulations of the hospital. The amount agreed upon for

the maintenance of such insane persons in a State hospital shall be secured by a properly executed bond, and bills therefor shall be collected monthly.

§ 70. **Entries in Case Book.** Every superintendent or other person in charge of an institution for the care and treatment of the insane, shall, within three days after the reception of a patient, make, or cause to be made, a descriptive entry of such case in a book exclusively set apart for that purpose. He shall also make or cause to be made entries from time to time, of the mental state, bodily condition, and medical treatment of such patient during the time such patient remains under his care, and in the event of the discharge or death of such person he shall state in such case book the circumstances thereof, and make such other entries at such intervals of time and in such form as may be required by the commission.

§ 71. **Transfer of Patients when Hospital is Overcrowded.** When the building of any State hospital shall become overcrowded with patients, or the number of buildings shall be reduced by fire or other casualties, or for other cause, the commission may, in its discretion, cause the transfer of patients therefrom, or direct that patients required to be sent thereto be transferred to another State hospital, where they can be conveniently received, or make, in special emergencies, temporary provision for their care, preference to be given in such transfers to a hospital in an adjoining rather than in a remote district. The expenses of such transfer shall be chargeable to the State, and the bills for the same, when approved by the commission, shall be paid by the treasurer of the State, on the warrant of the comptroller, out of any moneys provided for the support of the insane.

§ 72. **Investigation into the Care and Treatment of the Insane.**—When the commission has reason to believe that any person adjudged insane is wrongfully deprived of his liberty, or is cruelly, negligently or improperly treated, or inadequate provision is made for his skilful medical care, proper supervision, and safekeeping, it may ascertain the facts, or may order an investigation of the facts by one of its members. It, or the commissioner conducting the proceeding, may issue compulsory process for the attendance of witnesses and the production of papers and exercise the powers conferred upon a referee in the Supreme Court. If the commission deem it proper, it may issue an order directed to any or all institutions, directing and providing for such remedy or treatment, or both, as shall be therein specified. If such order be just and reasonable, and be approved by a justice of the Supreme Court, who may require a notice to be given of the application for such approval, it shall be binding upon any and all institutions and persons to which it is directed, and any wilful disobedience of such order shall be a criminal contempt and punishable as such. Whenever the commission shall undertake an investigation into the general management and administration of any institution for the insane, it may give notice to the attorney-general of any such investigation, and the attorney-general shall appear personally or by deputy and examine witnesses who may be in attendance. The commission, or any member thereof, may at any time visit and examine the inmates of any county or city almshouse, to ascertain if insane persons are kept therein.

§ 73. **Habeas Corpus.**—Any one in custody as an insane person is entitled to a writ of habeas corpus, upon a proper application made by him or some friend in his behalf. Upon the return of such writ the fact of his insanity shall be inquired into and determined. The medical history of the patient, as it appears in the case book, shall be given in evidence, and the superintendent or medical officer in charge of the institution wherein such person is held in custody, and any proper person, shall be sworn touching the mental condition of such person.

§ 74. **Discharge of Patients.**—The superintendent of a State hospital, on filing his written certificate with the commission, may discharge any patient, except one held upon an order of a court or judge having criminal jurisdiction in an action or proceeding arising out of a criminal offence at any time, as follows:

1. A patient who, in his judgment, is recovered.

2. Any patient who is not recovered, but whose discharge, in the judgment of the superintendent, will not be detrimental to the public welfare or injurious to the patient; provided, however, that before making such certificate the superintendent shall satisfy himself, by sufficient proof, that friends or relatives of the patient are willing and financially able to receive and properly care for such patient after his discharge. When the superintendent is unwilling to certify to the discharge of an unrecovered patient upon request, and so certifies in writing, giving his reasons therefor, any judge of a court of record in the judicial district in which the hospital is situated may, upon such certificate and an opportunity of a hearing thereon being accorded the superintendent, and upon such other proofs as may be produced before him, direct, by order, the discharge of such patient, upon such security to the people of the State as he may require, for the good behavior and maintenance of the patient. The certificate and the proof and the order granted thereon shall be filed in the clerk's office of the county in which the hospital is situated, and a certified copy of the order in the hospital from which the patient is discharged. The superintendent may grant a parole to a patient not exceeding thirty days, under general conditions prescribed by the commission. The commission may, by order, discharge any patient in its judgment improperly detained in any institution. A poor and indigent patient discharged by the superintendent, because he is an idiot, or an epileptic, not insane, or because he is not a proper case for treatment within the meaning

of this chapter, shall be received and cared for, by the superintendent of the poor or other authority having similar powers, in the county from which he was committed. A patient, held upon an order of a court or a judge having criminal jurisdiction, in an action or proceeding arising from a criminal offence, may be discharged upon the superintendent's certificate of recovery, approved by any such court or judge.

§ 75. **Clothing and Money to be Furnished Discharged Patients.**—No patient shall be discharged from a State hospital without suitable clothing adapted to the season in which he is discharged; and if it can not be otherwise obtained, the steward shall, upon the order of the superintendent, furnish the same, and money not exceeding twenty-five dollars, to defray his necessary expenses until he can reach his relatives or friends, or find employment to earn a subsistence.

§ 76. **Transfer of Non-resident Patients.**—If an order be issued by any judge, committing to a State hospital a poor or indigent person, who has not acquired a legal settlement in this State, the Commission in Lunacy shall return such insane person, either before or after his admission to a State hospital, to the county or State to which he belongs, and for such purpose may expend so much of the money appropriated for the care of the insane as may be necessary, subject to the audit of the comptroller.

§ 77. **Insane Indians.**—Poor and indigent insane Indians living within this State or upon any of the Indian reservations shall be committed to, confined in, and discharged from the State hospitals for the insane in the same manner and under the same rules and regulations as other poor and indigent insane persons; and all the provisions of this chapter shall apply to the Indians residing within this State the same as to other persons.

ARTICLE IV.

STATE HOSPITALS FOR INSANE CRIMINALS.

SECTION 90.—**Establishment and Purposes of the Matteawan State Hospital.**—The grounds, buildings, and property located at Matteawan, in the county of Dutchess, and used for the purposes of the hospital for insane criminals, are hereby declared to be the Matteawan State Hospital, to be used for the purpose of holding in custody and caring for such insane persons as may be committed to the said institution by courts of criminal jurisdiction, and for such convicted persons who may be declared insane while undergoing sentence at any of the various penal institutions of the State.

§ 91. **Medical Superintendent.**—The Superintendent of State Prisons shall, whenever there is a vacancy, appoint a medical superintendent for the Matteawan State Hospital, who shall be a well-educated physician of at least five years' actual experience in a hospital for the care and treatment of the insane. The Superintendent of State Prisons, subject to the approval of the State Commission in Lunacy, shall make by-laws and regulations for the government of the hospital and the management of its affairs.

§ 92. **Medical Superintendent as Treasurer of the Hospital.**—The medical superintendent shall be the treasurer of the hospital, and before entering upon his duties shall file with the comptroller of the State his undertaking to the people with sureties to be approved by the Superintendent of State Prisons, to the effect that he will faithfully perform his trust as such treasurer. He shall have the custody of the moneys, securities, and obligations belonging to the hospital, and shall open with some bank, in the vicinity of the hospital, to be selected with the approval of the comptroller, an account in his name as such medical superintendent, and immediately deposit in such bank all moneys received by him as such medical superintendent and treasurer, and shall draw therefrom only for the use of the hospital and in the manner provided by the by-laws and upon the order of the steward, specifying the object of each payment. He shall keep a full and accurate account of the receipts and payments, as directed by the by-laws, and of such other matters as the Superintendent of State Prisons and the State Commission in Lunacy may prescribe, and balance all his accounts, annually, on the thirtieth day of September, and within ten days thereafter deliver to the Superintendent of State Prisons, a statement thereof and an abstract of such receipts and payments for the past year. His books and vouchers shall at all times be open to the inspection of the Superintendent of State Prisons and the commission, and they may at any time require of him a statement of his accounts and of the funds and property in his custody.

§ 93. **Salaries of Resident Officers.**—The Superintendent of State Prisons shall, from time to time, determine the annual salaries and allowances of the resident officers, provided they do not in the aggregate exceed twelve thousand dollars; and the same shall be paid quarterly on the last days of March, June, September, and December, by the treasurer of the State, on the warrant of the comptroller, out of any moneys in the treasury not otherwise appropriated, to the medical superintendent, on his presenting a bill of particulars thereof signed by the steward, and properly certified by such medical superintendent.

§ 94. **Powers and Duties of Medical Superintendent and Assistants.**—The medical superintendent shall be the chief executive officer of the hospital, and shall:

1. Have the general superintendence of the building and grounds, together with their furniture, fixtures, and stock, and the direction and control of all persons therein, subject to the rules and regulations adopted by the Superintendent of State Prisons, with power to assign their respective duties.

2. Appoint such number of assistant physicians, not to exceed one for each two hundred inmates or fraction thereof, as the necessities of the institution may require, also a steward and matron, all of whom and the medical superintendent shall reside in the hospital, and shall be known as the resident officers thereof.

3. Appoint such and so many attendants and other subordinate employés as he may think proper and necessary for the economical and efficient administration of the affairs of the hospital, and prescribe their several duties and places, and fix, with the approval of the Superintendent of State Prisons, their compensation, and discharge any of them at his sole discretion; but in every case of discharge, so occurring, he shall, forthwith, enter the same with the reasons therefor, under an appropriate heading, in one of the record books of the hospital.

4. Give, from time to time, such orders and instructions as he may deem best calculated to insure good conduct, fidelity, and economy in every department of labor and expense.

5. Maintain salutary discipline among all who are employed by the institution, and enforce strict compliance with all instructions and orders given by him, and uniform obedience to all the rules and regulations of the hospital.

6. Cause full and fair accounts and records of all his doings, and of the entire business and operations of the institution to be kept regularly, from day to day, in books provided for that purpose, in the manner and extent prescribed in the by-laws.

7. See that all accounts and records are fully made up to the last day of September in each year, and present the principal facts and results, with his report thereon, to the Superintendent of State Prisons, within forty days thereafter. The resident officers, before entering upon their duties as such, shall severally take and file in the office of the secretary of State the constitutional oath of office. The first assistant physician shall perform the duties and be subject to the responsibilities of the superintendent in his sickness or absence. The steward may personally purchase any supplies for the use of such hospital, but only in the name of the medical superintendent, and in each instance by his direction and not otherwise.

§ 95 **Monthly Estimates.**—The medical superintendent shall cause an estimate to be made monthly, in accordance with forms to be approved by the State comptroller, of all moneys necessary for the support and maintenance of the hospital, which may be required to supplement the deficiencies in the earnings thereof. Such estimate shall be submitted to and examined by the Superintendent of State Prisons, who, if he is satisfied that it is correct, and that the articles named therein are actually needed for the support and maintenance of the hospital, shall certify to the same, and on production of such estimate so certified, to the comptroller, he shall draw his warrant on the State treasurer for the amount thereof, and the State treasurer shall pay such amount to the medical superintendent of the hospital, out of any money in the treasury appropriated for the support of such hospital.

§ 96. **Power of Removal.** The Superintendent of State Prisons may remove the medical superintendent for cause shown, and an opportunity shall be given to such Superintendent to be heard thereon; and such officer shall not be reappointed to the office of medical superintendent or to any other position in said hospital.

§ 97. **Transfer of Insane Convicts to the Matteawan State Hospital.**—Whenever the physician of either of the State prisons, county penitentiaries, or of the State Reformatory or other penal institutions, shall report in writing to the warden or other officer in charge thereof, that any convict confined therein is, in his opinion, insane, such warden or other officer shall apply to a judge of a court of record to cause an examination to be made of such person by two legally qualified examiners in lunacy, other than a physician connected with such State prison, penitentiary, reformatory, or penal institution qualified to act as medical examiners in lunacy. Such examiners shall be designated by the judge to whom the application is made. Such examiners, if satisfied, after a personal examination, that such convict is insane, shall make a certificate to such effect in the form and manner prescribed by this chapter for the commitment of insane persons to State hospitals. Such warden or other person in charge shall apply to a judge of a court of record for an order transferring such convict to the Matteawan State Hospital, accompanying such application with such certificate in lunacy. Such judge, if satisfied that such convict is insane, shall issue such order of transfer, and such warden or other officer in charge shall thereupon cause such convict to be transferred to the Matteawan State Hospital, and delivered to the medical superintendent thereof. At the time of such transfer, the certificate in lunacy and order of transfer shall be presented to such medical superintendent, and a copy thereof shall be placed on file in the office of the Superintendent of State Prisons. Such insane convict shall be received into such hospital and detained there until legally discharged. Such warden, or other officer in charge, before transferring such insane convict, shall see that he is bodily clean, and is provided with a new suit of clothing similar to that furnished to convicts on their discharge from prison. The costs necessarily incurred in determining the question of insanity, including the fees of the medical examiners, shall be a charge upon the State or the municipality at whose expense the institution from which the transfer is made or sought to be made is maintained.

§ 98. **Disposal of Insane Convicts after Expiration of Term of Imprisonment.**—Whenever any convict in the Matteawan State Hospital, under and by virtue of this Act, shall

continue to be insane at the expiration of the term for which he was sentenced, he may be retained therein until he has recovered or is otherwise legally discharged. The medical superintendent of such hospital may discharge and deliver any patient whose sentence has expired and who is still insane, but who, in the opinion of the superintendent is reasonably safe to be at large, to his relatives or friends who are able and willing to comfortably maintain him, without further public charge; and such patient may, in the discretion of the medical superintendent, be provided with the whole or a portion of such allowances as are hereinafter granted to recovered convicts. Whenever any convict, who, by reason of his insanity, shall have been retained beyond the date of the expiration of his sentence shall recover, he may be discharged by the medical superintendent, and such convict shall be entitled to ten dollars in money, suitable clothing, and a railroad ticket to the county of his conviction or to such other place as he may designate at no greater distance. Similar allowances shall be made to patients committed by order of a court and who may be discharged. Any convict in the Matteawan State Hospital, whose term of imprisonment has expired by commutation or otherwise, and who is not recovered may, upon an order of the Commission in Lunacy, be transferred to any institution for the insane.

§ 99. **Convicts on Recovery to be Transferred to Prison.**—Whenever any convict, who shall have been confined in such hospital as an insane person, shall have recovered before the expiration of his sentence, and the medical superintendent thereof shall so certify in writing to the agent and warden or other officer in charge of the institution from which such convict was received or to which the Superintendent of State Prisons may direct that he be transferred, such convict shall forthwith be transferred to the institution from which he came by the medical superintendent of the hospital, or if received from one of the State prisons, to such State prison as the Superintendent of State Prisons may direct; and the agent and warden shall receive such convict into such institution, and shall, in all respects, treat him as when originally sentenced to imprisonment. Any inmate not a convict, held upon an order of a judge, in a criminal proceeding, may be discharged therefrom upon the superintendent's certificate of recovery, made to and approved by such court or judge.

§ 100. **Certificate of Conviction to be Delivered to Medical Superintendent and Copy Filed.**—Whenever any convict shall be transferred to the Matteawan State Hospital, the agent and warden or other officer in charge of the prison, penitentiary, reformatory, or other penal institution from which such convict is transferred, shall cause a correct copy of the original certificate of conviction of such convict to be filed in the office of the warden or officer in charge, and shall deliver the original certificate to the medical superintendent of such hospital; and whenever any such convict shall be transferred to any penal institution from such hospital, as hereinbefore provided, the medical superintendent shall deliver to the agent and warden, or other officer in charge of such institution, such original certificate, which shall be filed in the clerk's office of the same.

§ 101. **Transfer from State Hospitals to Matteawan State Hospital.**—The Commission in Lunacy may, by order in writing, transfer any insane inmate of a State hospital, committed thereto upon the order of a court of criminal jurisdiction, to the Matteawan State Hospital, and the county in which the criminal charge arose or conviction or acquittal was had, if the person was then a resident of that county, shall defray all the expenses of such person while at the Matteawan State Hospital, and the expenses of returning him to such county or from such hospital to one of the other State hospitals; in any other case such expenses shall be a charge against the State.

§ 102. **Authority to Recover for the Support of Patients.**—The medical superintendent of the hospital is hereby authorized to recover for the support of any patient therein, chargeable under the law to either counties or penitentiaries, in an action to be brought, in the name of the people of the State of New York, against the county or penitentiary, for the maintenance of said patient.

§ 103. **Tenure of Office.**—Nothing in this article shall be construed to affect the tenure of office of any of the present officers of the hospital.

§ 104. **Communications with Patients.**—No person not authorized by law or by written permission from the Superintendent of State Prisons shall visit the Matteawan State Hospital, or communicate with any patient therein without the consent of the medical superintendent, nor without such consent shall any person bring into or convey out of the Matteawan State Hospital any letter or writing to or from any patient; nor shall any letter or writing be delivered to a patient, or if written by a patient be sent from the Matteawan State Hospital until the same shall have been examined and read by the medical superintendent or some other officer of the hospital duly authorized by the medical superintendent. But communications addressed by such patient to the county judge or the district attorney of the county from which he was sentenced, shall be forwarded, after examination by such medical superintendent, to their destination.

SECTION 113, CHAPTER 546, LAWS 1896.

ADMISSION OF INSANE PATIENTS FROM CRAIG COLONY.

Discharge of Patients.— * * * Should an epileptic become insane, such patient, if a State patient, shall be sent to the State hospital of the district of which he was a resident just prior to his admission to the colony in the manner prescribed by law. The bills for the reasonable expenses incurred in the transportation of State patients to and from the State hospitals after they have been approved in writing by the State Commission in Lunacy, shall be paid by the treasurer of the State on the warrant of the comptroller from the funds provided for the support of the State hospitals. In case the relatives, guardians, or friends of such an insane patient desire that he become an inmate of any State hospital situated beyond the limits of the district of which he was formerly a resident, and there be sufficient accommodations in such State hospital, he shall be received there in the manner provided by law for the transfer of other insane persons. Private patients, who may become insane, shall be committed, as prescribed by law, subject to the regulations of the State commission in lunacy, to such institution for the insane as may be designated by the relatives, guardians, or friends of such insane person, all travelling and other expenses of removal to be paid by them. After any patient has been delivered to the managers or officers of such hospital or institution, the care and custody of the managers of the colony over such insane person shall cease; and after any patient shall, as aforesaid, be so certified to be insane as prescribed by law, such patient shall come under the supervision of the State Commission in Lunacy.

NORTH CAROLINA.

(State Hospital, Raleigh; State Hospital, Morganton; State Hospital, Goldsboro.)

Inquisition of Lunacy.—On petition to the Superior Court of the county where the alleged idiot, inebriate, or lunatic resides, an inquisition may be had by a jury of twelve men. If such person be found an idiot, inebriate, or lunatic, a guardian may be appointed as in cases of orphans. (Code of North Carolina, § 1670.)

Appointment of Guardian for Lunatic Confined in Asylum.—If any person be confined in any asylum for lunatics and insane persons, the certificate of the superintendent declaring such person to be of unsound mind and memory, sworn to and subscribed before the clerk of the Superior Court of the county in which such asylum is situated, shall be sufficient evidence to authorize the clerk to appoint a guardian for such person. (Idem, § 1673.)

Sale of Property of Insane Person.—Property of an insane person may be sold on the order of the clerk of the Superior Court, when it appears to him that the personal estate has been expended, or is insufficient for the support of the lunatic, and that he is likely to become a charge upon the county. The order shall specify the property to be disposed of. Such order shall be made upon the petition of the guardian setting forth that such sale is necessary for the support of the insane person, or for the discharge of debts incurred for his maintenance. (Idem, §§ 1674, 1675.)

Surplus Income.—Whenever the annual income of the estate of a lunatic is more than sufficient to support himself and to maintain and educate the members of his family, the clerk of the court may direct that fit and proper advancements be made out of the surplus of such income to children and grandchildren, not being members of his family and entitled to be supported, educated, and maintained out of such estate. Such advancements shall only be made for the better promotion in life of such as are of age or married, and for the maintenance, support, and education of such as are under the age of twenty-one years and unmarried. In every application for such advancement the guardian of the insane person and of persons entitled to a distributive share of the estate shall be made parties. Such advancement shall be made in the same equal manner as if made by the insane person himself, and every sum advanced to a child or grandchild shall be an

advancement, and shall bear interest from the time it was received. The clerk may select and decree advancement to such as most need the same. The clerk shall withhold advancements from such persons as will probably waste them. (Idem, §§ 1677–1682.)

State Asylums.—There are three hospitals for the insane maintained at the expense of the State. Two are for the accommodation of the white insane, and the State Hospital at Goldsboro is exclusively for the colored insane of the State. Each hospital is under the management of a board of nine directors holding office for six years. Three members of the board constitute an executive committee, who hold office for a term of one year and perform such duties as are delegated by the board. Each board directs and manages the affairs of the institution under its charge, and may appoint a superintendent thereof and prescribe his duties. The superintendent shall be a skilful physician, educated to his profession, of good moral character, of prompt business habits, and of kindly disposition. He shall hold office for a term of six years unless sooner removed by the board for unfaithfulness to his trust, gross immorality, or incompetency. Each board also appoints one or more assistant physicians. Such assistants may be suspended by the superintendent, with the advice and consent of the executive committee, for insubordination, immorality, neglect of duty, or incompetency, until a meeting of the board of directors, to be called within thirty days, when the charges against such physicians shall be tried and determined. Stewards and matrons are appointed by the directors on the nomination of the superintendent. The State treasurer is treasurer of the hospital. Each board makes such by-laws and regulations for the government of the institution under its charge as may be necessary. (Laws of 1899, ch. 1, §§ 11–4.)

Admission into State Asylums.—An affidavit shall be filed in writing with the clerk of the Superior Court of the county by any person requesting the examination of the insane person setting forth the belief that he is insane and a fit subject for admission into an insane asylum. Unless the person in whose care the alleged insane person is will agree to bring him before said clerk, or unless the clerk is of opinion that it will be injurious to the insane person to be brought before him, the clerk shall issue his precept directing the sheriff or other lawful officer to bring such insane person before him. Upon the appearance of the insane person the clerk shall call to his assistance the county physician or some other reputable physician, and shall proceed to examine into the condition of the alleged insane person. If he decides that the person is sane, he shall discharge him; if insane, he shall direct a warrant to the sheriff, committing the person to the proper hospital. The clerk is required to keep a record of all examinations. A person shall not be committed unless he is a *bona fide* resident of the State. No cretin or idiot shall be committed. The questions to be answered by the examining physician and witnesses are prescribed by statute, and the answers thereto are to be written and subscribed and sworn to by the witness. The superintendent, if doubtful as to whether a patient should be admitted, may convene three members of the board of directors for the purpose of examining and deciding whether such patient is a proper subject for admission. (Idem, §§ 15–21.)

NORTH DAKOTA.

(State Hospital for the Insane of North Dakota, Jamestown.)

The term insane includes any species of insanity or mental derangement. The term idiot is restricted to persons supposed to be without mind. No idiot shall be admitted into any hospital for the insane. (Revised Codes of North Dakota, § 1532.)

Guardians.—The County Court, on the verified petition of any relative or friend that any person is insane, or from any cause is mentally incompetent to manage his property, shall cause such person to be cited. If, after a full hearing and examination, it appears to the court that the person in question is incapable of taking care of himself and managing his property, he shall appoint a guardian of his person and estate. (Idem, §§ 6549, 6550.)

Powers of Guardian.—Every guardian appointed as provided in the preceding section has the care and custody of the person of his ward and the management of all his estate until he is legally discharged. The powers and duties of guardians of insane persons are similar in all respects to those of guardians of minors as prescribed in the Probate Code, Chapter 7. (Idem, § 6551.)

Restoration of Insane.—Any person who has been declared insane, or the guardian or relatives or friends of such person, may apply by petition to the County Court of the county in which he was declared insane, to have the fact of his restoration to capacity judicially determined. Notice of a hearing shall be given to the guardian of the petitioner, to the husband or wife, if there be one, and to the father or mother, if living in the county. On the trial the guardian or relative, and in the discretion of the judge any other person, may contest the right of the petitioner to the relief demanded. Witnesses may be required to appear and testify. If it be found that the petitioner is of sound mind and capable of taking care of himself and of his property, his restoration to capacity shall be adjudged and the guardianship shall cease. (Idem, § 6552.)

Commissioners of Insanity.—There is in each county a board of commissioners, consisting of three persons, known as Commissioners of Insanity. The county judge is a member and chairman of such board; one of them shall be a lawyer and one a physician. Such commissioners have cognizance of all applications for admission to the hospital, or for the safekeeping of insane persons within their county. Application for admission to the hospital shall be made in writing in the nature of an information alleging that the person on whose behalf the application is made is believed to be insane and a fit subject for custody and treatment in the hospital. The grounds of the information shall thereupon be investigated by the commissioners. They may require that the person be brought before them and examined, and may issue their warrant therefor. They shall hear testimony for and against the application. Any citizen or relative of the insane person may appear by counsel, if they so elect. The commissioners shall appoint some regularly practising physician of the county to make a personal examination touching the truth of the allegations in the information, and the actual condition of such person, and report to them thereon. On the return of the physician's certificate the commissioners shall conclude their investigation and find whether the person is insane and a fit subject for treatment and custody in the hospital. If he is found not insane, they shall order his discharge. If insane, they shall issue their warrant authorizing the superintendent of the

hospital to receive such person as a patient. If such person cannot be admitted into the hospital and cannot with safety be allowed to go at liberty, the commissioners shall require him to be suitably provided for otherwise until such admission can be had. Such patients may be cared for either as public or private patients. Private patients are those whose relatives or friends obligate themselves to take care of and provide for them without public charge. The questions to be answered by the examining physician are prescribed by statute. (Idem, §§ 1513–1526.)

Writ of Habeas Corpus.—All persons confined as insane shall be entitled to the benefit of habeas corpus, and the question of insanity shall be decided at the hearing; and if the judge or court shall decide that the person is insane, such decision shall be no bar to the issuing of a writ the second time whenever it shall be alleged that such person has been restored to reason. (Idem, § 1527.)

Postal Rights of Insane Persons.—Each inmate of all insane asylums shall be allowed to choose one individual from the outside world to whom he may write when or whatever he desires, and over these letters there shall be no censorship, and his post-office rights, so far as this one individual is concerned, shall be as free and unrestricted as those of any other resident or citizen. Such inmates shall be furnished with suitable material for writing, and closing, sealing, stamping, and mailing letters sufficient for the writing of at least one letter every week. (Idem, § 1533.)

State Hospital.—As to the care and management of such hospital, see Article 8, of Chapter 10, of the North Dakota Political Code.

OHIO.

(Dayton State Hospital, Dayton; Columbus State Hospital, Columbus; Athens State Hospital, Athens; Cleveland State Hospital, Cleveland; Toledo State Hospital, Toledo; Massillon State Hospital, Massillon; Long View Hospital, Carthage.)

The terms insane and lunatic include every species of insanity or mental derangement; the term idiot is restricted to a person foolish from birth—one supposed to be actually without mind. (Statutes of Ohio, Giauque ed., 1894, § 720.)

Guardians of Lunatics, Idiots, and Imbeciles.—The Probate Court, upon satisfactory proof that any person is a lunatic, idiot, or imbecile, shall appoint a guardian for such person. No such guardian shall be appointed except upon three days' notice to the person's next of kin residing in the county. If the wife of such person is competent, the probate judge may appoint her as his guardian. All laws relating to guardians for minors and their wards, and pointing out the duties, rights, and liabilities of such guardians and their sureties, are applicable to such guardians. In the settlement of accounts of guardians of idiots, imbeciles, or lunatics, no voucher shall be received from or allowed as a credit to the guardian which is signed by such idiot, imbecile, or lunatic. (Idem, §§ 6302–6304.)

Management of Real Estate of Ward.—Whenever the sale of the real estate of a ward is necessary for his support, or the support of his family, or the payment of his debts, or when such sale will be for the interest of such ward or his children, the guardian may sell the same under like proceedings as are required to authorize the sale of real estate by the guardian of a minor. But if it be more for the interest of such ward or his children, the Probate Court upon the petition of the guardian may authorize and sell all such real estate in private. The guardian may in the same manner as the guardian of a minor be authorized to lease and improve the real estate of his

ward; and if the lease extend beyond the time of the restoration of such ward to sound mind or his death, such lease shall terminate on his restoration or death, unless such lease be confirmed by such ward or his legal representatives; but in case of such termination of the lease the tenant shall have a lien upon the premises for any sum expended by him in making improvements. The Probate Court may authorize the guardian to lease the real estate for a term of years or by perpetual lease, with or without the privilege of purchase if it is necessary for the support of the ward or his family, or if such lease will be for the best interest of him or them. The application for authority to make such a long lease is by petition setting forth the necessary facts. On filing the petition the same proceedings shall be had as on petition for sale of the real estate of a minor. The court may prescribe the terms, covenants, conditions, and stipulations of the lease. (Idem, §§ 6306–6313.)

Insolvency of Lunatic.—If the estate of an idiot, imbecile, or lunatic is insolvent, the same shall be settled in like manner and like proceedings may be had as are required by law for the settlement of the insolvent estate of a deceased person. (Idem, § 6314.)

Guardians of Drunkards.—The Probate Court, upon satisfactory proof that any person is incapable of taking proper care of himself or his property by reason of intemperance or habitual drunkenness, shall appoint a guardian of the person and property of such person; and all laws relating to guardians for lunatics, idiots, or imbeciles, and their wards, are applicable to such guardians. (Idem, § 6317.)

Commitment of the Insane to State Hospitals.—The State of Ohio is divided into districts, in each of which is situated a State hospital for the insane. Each county is entitled to send patients to the hospital of the district in which such county is situated in proportion to the population of such county. The medical superintendent of each of the hospitals shall inform the probate judge of each of the counties in such hospital district monthly of the quota of patients to which each county is entitled and the number in the hospital from such county. Patients are admitted on the filing of an affidavit of some resident citizen of the county with the probate judge, alleging that such person is insane, and that because thereof his being at large is dangerous to the community. The probate judge shall cause such alleged insane person to be apprehended and brought before him upon a certain day, at which time, if any person disputes the insanity, the probate judge shall issue subpœnas for such persons as are required to be examined on behalf of the alleged insane person. The judge shall proceed to examine the witnesses and attendants; and if he is satisfied that the person is insane, he shall cause a certificate to be made out by the medical witness and attendants, setting forth such facts as are prescribed by the statute. Upon receiving such certificate he shall forthwith apply to the superintendent of the hospital situated in the district in which such patient resides, and at the same time transmit copies of the medical witnesses' certificates; and if the probate judge is advised that the medical superintendent will receive the patient, he shall cause him to be sent to the asylum. When the patient is sent to the asylum, the probate judge shall see that he is supplied with the proper clothing; and if not otherwise furnished, he shall furnish such clothing, which shall be a county charge. If the person found to be insane cannot be admitted to the asylum, the probate judge shall cause such person to be properly cared for. (Idem, §§ 698–707, as amended by Laws of 1894, pp. 23, 24.)

OREGON.

(Oregon State Insane Asylum, Salem.)

The words "insane person" are intended to include every idiot, every person not of sound mind, every lunatic and distracted person; and the word "spendthrift" is intended to include any one who is liable to be put under guardianship on account of excessive drinking, gaming, idleness, or debauchery. (Hill's Annotated Laws, § 2911.)

Jurisdiction.—The County Court has exclusive jurisdiction in the first instance to take the care and custody of the person and estate of a lunatic or habitual drunkard, and to appoint and remove guardians therefor; to direct and control the conduct of such guardians, and to settle their accounts. (Idem, § 895.)

Guardians.—Guardians of insane persons are appointed by the County Court upon application of relatives or friends of such insane person, or of any other person residing in the county where such insane person resides. The judge shall cause a notice to be given to the person alleged to be insane of the time and place appointed for the hearing, not less than ten days before such time. If, after a full hearing, it shall appear to the judge that the person in question is incapable of taking care of himself, the judge shall appoint a guardian of his person and estate. (Idem, § 2889.)

Powers and Duties.—The guardian so appointed has the care and custody of the person and the management of the estate of such insane person until he shall be legally discharged, and he shall give a bond to the State of Oregon in like manner as guardians of minors. (Idem, § 2890.)

Guardians for Spendthrifts.—When any person by excessive drinking, gaming, idleness, or debauchery of any kind so spends, wastes, or lessens his estate as to expose himself or family to want or suffering, or the county to expense for the care of himself or his family, the County Court shall present a complaint to the county judge setting forth the facts and circumstances of the case, and praying to have a guardian appointed for him. Notice shall be given to such supposed spendthrift of the time and place of the hearing, not less than ten days before such hearing; and if after a full hearing it shall appear that the person complained of comes within the description, the judge shall appoint a guardian of his person and estate. A copy of the complaint shall be filed in the office of the county clerk after the order of notice has been issued. When a guardian shall be appointed for the insane person or spendthrift, the judge shall make an allowance to be paid to the guardian for all reasonable expenses incurred by the ward in defending himself against the complaint. The guardian so appointed has the care and custody of the person and management of the estate in the same manner as guardians of insane persons. (Idem, §§ 2891–2895.)

Management of Estate.—The guardian shall apply the income and profits of the ward's estate for the comfortable and suitable maintenance and support of the ward and his family. If they be insufficient for that purpose, the guardian may sell the real estate upon obtaining a license therefor as provided by law. The estate of the ward shall be appraised by three suitable persons, appointed and sworn as required with respect to the inventory of the property of a deceased person. The county courts upon application of the guardian or any person interested in the estate of the ward may authorize the transfer and reinvestment of the property of the ward. (Idem, §§ 2897–2899.)

Commitment of Insane.—The county judge, upon application of any citizen in writing setting forth that any person, by reason of insanity or idiocy, is suffering from neglect, exposure, or otherwise, or is unsafe to be at large, shall cause such person to be brought before him, and at the same time and place one or more competent physicians, who shall examine the person alleged to be insane or idiotic. If such physician or physicians, after careful examination, shall certify upon oath that such person is insane or idiotic, then the judge, if in his opinion such person be insane or idiotic, shall cause such person to be placed in the insane asylum of the State of Oregon. An appeal shall lie in the County Court in such case in the same manner as in all other cases. But no insane or idiotic person shall be committed to the asylum who has friends desiring to provide for his safekeeping and medical treatment. (Idem, § 3557, as amended by Laws of 1891, p. 112.)

All the proceedings upon such application and the judgment of the court shall be recorded in the records of the County Court. When the patient is adjudged insane, the county judge shall make a warrant reciting his findings, the cause of the insanity where the same can be ascertained, together with the name, age, nativity, and present residence of the patient. The expense of sending insane and idiotic persons committed to the asylum shall be paid by the State treasurer out of the fund appropriated for such purpose. The cost of examination and committal shall first be paid by the county, and afterward repaid by the State treasurer upon the certificate of the county judge and the audit of the secretary of State by the State treasurer, out of funds appropriated for that purpose. (Idem, § 3558.)

Oregon State Insane Asylum.—The State Insane Asylum is governed by a board of trustees composed of the governor, secretary of State, and the State treasurer. They appoint all officers and employés of the asylum, prescribe their duties, and remove them when in their judgment the good of the public service requires it. They are required to visit the asylum once in three months, and to keep themselves constantly advised of all items of labor and expense, and the condition of the buildings and property of the asylum. They are required to report biennially to the legislative assembly. They shall appoint a medical superintendent, who shall serve four years or during good behavior, and on his nomination one or two assistant physicians and other officers according to the requirements of the institution. The superintendent and all the assistant physicians shall reside at the asylum, and shall be regular graduates in medicine. The superintendent is the executive officer of the asylum under the regulations and by-laws of the board of trustees. He has control of the patients, prescribes and directs their treatment, adopts sanitary measures for their welfare, and discharges such as in his opinion have permanently recovered their reason, or such other patient as the best interests of the State and the institution require. (Idem, §§ 3549–3553.)

Death and Discharge of Patients.—In case of the death or discharge of a patient committed to the State Asylum, the superintendent shall notify the County Court of the county from which the patient was committed of such death or discharge and the date thereof. The County Court upon receipt of such notice is required to enter an order showing such death or discharge. If a patient is reported discharged as cured, the entry of such order is *prima facie* evidence of the removal of all legal disability arising from an adjudication of insanity. (Laws of Oregon, 1895, p. 41.)

PENNSYLVANIA.

(State Hospital for Insane, Norristown; Pennsylvania State Hospital, Harrisburg; Philadelphia Hospital—Insane Department, Philadelphia; State Hospital for the Insane, Warren; State Hospital for the Insane, Danville; Asylum for the Chronic Insane, Wernersville.)

Commission to Inquire into Lunacy or Habitual Drunkenness.—Jurisdiction to issue a commission in the nature of a writ *de lunatico inquirendo* to inquire into lunacy or habitual drunkenness of any person is vested in any Court of Common Pleas. It is to be issued by the court of the county in which the person resides. The form of the commission is prescribed by statute. No commission shall be issued, except upon application in writing of a relative by blood or marriage of the person therein named, nor unless such application be accompanied by affidavits of the truth of the facts therein stated. If the alleged lunatic or habitual drunkard has no such relative, any disinterested person of the same township, ward, or borough may make application to the court for such a commission. The commission may be directed to any one or more persons. Upon granting the application for a commission the court shall give such notice to the alleged lunatic or habitual drunkard or his near relatives or friends as it shall deem advisable. The commissioner or commissioners shall summon such number of persons, not less than six nor more than twelve, to attend upon the inquest as the circumstances of the case may seem to them to require. If upon such inquisition it be found that the party is not a lunatic or habitual drunkard, and that there was no cause for such application, the judge shall certify the same on such inquisition, and thereupon the party making the application shall be liable for the costs. Whenever any person shall be found to be insane, the committee of the person or of the estate, and also the clerk of the court, shall forthwith send to the committee on lunacy, at their office, the statement in writing of the name, age, sex, and residence of the lunatic, and the residence of the committee. The committee on lunacy, or any one or more of the members thereof, may visit and examine the said lunatic, or authorize such visit and examination, and may apply to any court having jurisdiction over the committee, or to the judge of the Court of Common Pleas, to make such orders for the maintenance, custody, and care of the lunatic, and for the care and disposition of his property, as the case may require. (Brightly's Purdon's Digest, pp. 1270–1272.)

Appointment of the Committee.—On the return of an inquisition finding the person a lunatic or habitual drunkard, the court may commit the custody and care of the person or estate, or of both, to such person or persons as it may deem most suitable. A bond shall be given by the person appointed committee of the estate in such sum as the court shall direct, with condition for the faithful performance of the trust and for a due account of all property and funds coming into his hands. (Idem, p. 1272.)

Powers and Duties of the Committee.—The committee, within forty days after undertaking the trust, shall file in the office of the prothonotary of the court a just and true inventory of all personal estate belonging to his ward, together with a statement of the real estate. The committee of the estate of every person found to be a lunatic or habitual drunkard shall have the management of the real and personal estate, and apply so much of the income as is necessary to the payment of the debts and for the support and maintenance of the person and of his family, and for the education of his minor children. If the income is not sufficient, under the direction of the

court the committee may apply so much of the principal of the personal estate as is necessary. Under the direction of the court the committee may invest the money of the ward in such stocks or securities as are approved by the court. Each committee shall account once in three years for the property committed to him. (Idem, p. 1273.)

Sale of the Real Estate of a Lunatic.—If the personal estate of a lunatic is not sufficient for the support of the lunatic, the court may authorize the sale of his real estate, upon an application by the committee setting forth a statement or inventory of the real and personal estate, the debts due by the lunatic or habitual drunkard, and an estimate of the amount properly required annually for his support and maintenance, and that of his family and the education of his children. No order shall be granted except upon due notice to the next of kin of the lunatic or habitual drunkard. Each order for the sale of real estate shall specify the property to be sold, the notice of the sale to be given by the committee, the terms of sale, the amount of security to be given by the committee, and the day on which the order is returnable. Each order for the mortgaging of real estate shall specify the amount to be raised, the property to be mortgaged, the rate of interest, the amount of security to be given by the committee, and the day on which the order is returnable. No sale or mortgage shall be confirmed by the court until the committee has given security for the faithful application of the proceeds. If the sale or mortgage be confirmed by the court, the committee shall execute such sale or mortgage according to the terms of the contract. On the application of the committee the court may authorize the sale of timber standing upon the lands of such lunatic or drunkard. (Idem, pp. 1274, 1275.)

Lunatic Asylums.—The Board of Public Charities has the supervision of all houses or places in which any person of unsound mind is detained. The board shall appoint a committee of five to act as the committee on lunacy. The two professional members, one a physician and the other a lawyer, shall be members of that committee. The committee on lunacy shall examine and report annually to the board the condition of the insane in the State, and the management and conduct of the hospitals, public and private, almshouses, and all other places in which the insane are kept for care and treatment or detention. The board shall have power to ordain rules and regulations relating to the licensing of all places where persons are detained as lunatics or of unsound mind, and for the insuring of proper treatment of persons so detained, and as to the forms to be observed in the commitment, transfer of custody, and discharge of all lunatics other than those committed by order of a court of record. (Idem, p. 1254.)

Admission to Insane Asylums.—No person shall be received as a patient in any insane asylum without a certificate signed by at least two physicians that they have examined separately the person alleged to be insane, and thoroughly believe that the person is insane, and that the disease is of a character requiring that the person should be placed in a hospital or other establishment where the insane are detained for care and treatment, and that they are not related by blood or marriage to the person alleged to be insane, nor in any way connected with the hospital or other establishment to which the patient is to be committed. There shall be delivered at the time of the admission of the patient a written statement signed by the person at whose instance the insane person has been removed and detained, containing the name, age, residence, occupation, parents, if living, husband or wife, children, brothers and sisters, and residence of each of these

persons; if not more than one of these classes is known, the names and residence of such of the next degree of relatives as are known; a statement of the time during which the insanity has been supposed to have existed, and the circumstances that induced the belief that insanity exists; and the name and address of all medical attendants of the patient during the past two years. A certificate of the physician and a statement furnished at the time of the reception of the patient shall be forwarded by mail to the committee on lunacy within seven days from such reception. Any physician designated by the lunatic, or any member of his family or near friend, shall be permitted at all reasonable hours to visit and examine the patient. All patients shall be given reasonable opportunity and furnished with materials for communicating with any person without the building. They shall have the unrestricted privilege of addressing communications, not oftener than once a month, to any member of the committee on lunacy. (Idem, pp. 1256, 1257.)

Commitment of Insane Persons by the Courts.—Insane persons may be placed in hospitals for the insane by the order of any court, and a statement in writing of any respectable citizen that a certain person is insane, and that his welfare or that of others requires his restraint. The judge shall thereupon appoint immediately a commission to inquire into the report upon the facts of the case. The commission shall be composed of three persons, one a physician and one a lawyer. In their inquisition they shall hear such evidence touching the merits of the case, as well as the statements of the party complained of. If in their opinion it is a suitable case for confinement, the judge shall issue his warrant for such disposition of the insane person as will promote the object desired. If the commission report that it is not a suitable case for confinement, the petitioner shall be liable for all costs. If the commission report the case a suitable one for confinement, and it shall appear that the lunatic has real or personal property, such property shall be liable for all costs. (Idem, p. 1253.)

Support of Indigent Insane.—Where a person is committed to a hospital for the insane by a judge or court in any county, the county from which the person is sent is liable to the hospital for his support. The county may recover from persons or poor district chargeable with such support. (Pennsylvania Laws of 1895, No. 184.)

Return of Indigent Insane to other States.—In all cases of the commitment of indigent insane, the court shall determine the legal residence of the insane person. If it be ascertained that the person committed is not a resident of the State, the clerk of the court shall notify the State Board of Charities. The State Board is required to investigate the question of residence, and if it is found that the patient is a resident of another State or country, the board is authorized to return him to such State or country. (Pennsylvania Laws of 1897, Act No. 145.)

RHODE ISLAND.

(State Hospital for the Insane, Cranston, Howard P. O.)

Restraint of Insane Persons.—Whenever complaint in writing and under oath shall be made to any justice or clerk of the District Court that any person within the county is insane so as to be dangerous to the peace or safety of the people of the State, or so as to render his restraint and treatment necessary for his own welfare, such justice or clerk shall cause such person

to be arrested and brought before some District Court for examination relative to such complaint. When the insane person cannot be examined in open court, such examination may be held at such times and places as shall be most conducive to the health and comfort of the person to be examined. If the court on such examination adjudge such complaint to be true, it shall commit such person to the Butler Hospital for the Insane or to the State Asylum for the Insane, to be detained until upon instruction and examination he shall be declared to be restored to soundness of mind. The warrant of commitment shall state the town in which such lunatic or mad person was arrested. The costs shall be paid out of the property of the lunatic if he have any, otherwise in the first instance by the State until the liability of some town in the State for the maintenance of such person is established. Commissioners may be appointed on petition under oath to inquire into the condition of the insane person, and to report all facts connected with the case, together with their opinion whether such person if insane should be placed in such hospital or State asylum. (Laws of 1893, ch. 1199, §§ 1–9.)

Examination of Confined Insane Person.—On petition of any person confined in an insane asylum, or of any person on his behalf, to a justice of the Supreme Court, setting forth that such person confined therein is not insane and is unjustly deprived of his liberty, such justice shall issue a like commission as provided for the commitment of the insane person, for the purpose of inquiring into the condition of such person. The person confined as insane shall have the right to confer with counsel, to produce evidence, and to be present at the inquisition. The petitioner or his counsel may examine the insane person at the place where he is confined. The commissioners shall make a personal examination of such insane person at the place where confined, without the presence of the superintendent or any other person connected with the institution; but no person detained as insane shall be taken from the institution without an order of the Supreme Court. Justices of the Supreme Court may either confirm or disallow the report of the commissioners, and order the recommitment or discharge of such person, or dismiss the petition altogether, as the facts may seem to require. (Idem, §§ 15–18.)

Appointment of Guardian.—Whenever any idiot or lunatic or person of unsound mind, or any person who from excessive drinking, gaming, idleness, or debauchery of any kind, or from want of discretion in managing his estate, shall be likely to bring himself or family to want, or to render himself or family chargeable, the Court of Probate shall have the right to appoint a guardian of the person and estate of such person. The guardian of any habitual drunkard shall have the right to commit the ward to any curative hospital, either within or without this State, until he is cured of his drunkenness, but not exceeding six months at any one time. The estate of the ward shall be chargeable with the expenses incident to such committal and custody. The provisions in regard to the guardianship of minors are also applicable to the guardianship of insane persons and habitual drunkards. (Public Statutes of Rhode Island, ch. 168.)

Inmates of State Hospital may Leave Temporarily.—The superintendent of the State Hospital for the Insane, acting under the authority of the Board of State Charities and Correction, may permit any of the inmates of the State Hospital to leave the hospital, temporarily, under the charge of relatives, friends, or guardians, for a period not exceeding sixty days. (Laws of 1898, ch. 576.)

SOUTH CAROLINA.

(State Hospital for the Insane, Columbia.)

Governing Body.—The governor appoints five regents of the State Hospital for the Insane, who are empowered to make and establish rules and regulations and by-laws for the government of the institution, and who report to the legislature. (Revised Statutes of South Carolina, §§ 2247, 2248.)

Patients are received on papers from the probate judge of the county in which the insane person resides. Only persons who are dangerously and permanently insane, or who are incompetent to protect their persons and property, are admissible. (Idem, § 2249.)

Commitment.—Application is made to the judge of the Court of Probate of the county in which the insane person is a resident, and the judge then proceeds to examine witnesses or not, in his discretion; and he reports his finding as to insanity to the superintendent of the hospital, recommending admission if insanity be found. Blanks are furnished by the regents containing interrogatories, which are required to be answered and filled in by the judge and forwarded to the board of regents. The superintendent replies to the report of the judge, whether or not the patient may be received. The superintendent may report the question of admisison to the board of regents. The probate judge, on the return of the superintendent, calls in two physicians, who certify as to the question of insanity. The judge then issues a certificate as to insanity and sends the patient with a certified copy of the same to the superintendent. The superintendent may keep in custody five days a person violently insane, even though no order is made, upon the certificates of two physicians. A bond is furnished that the usual order will be obtained. (Idem, §§ 2251–2253, and 2255.)

Upon the examination before the judge evidence is taken as to the property of the incompetent, to determine whether or not the patient shall be a paying one. Paying patients may be admitted on voluntary application when there is room for them. (Idem, § 2255.)

Whenever a patient is recovered, it becomes at once the duty of the board of regents to discharge him, and the board of regents are required to make to the legislature annually a full and complete report of the condition of the hospital as to accommodations, disbursements, treatment, etc. (Idem, §§ 2267, 2270.)

SOUTH DAKOTA.

(South Dakota Hospital for the Insane, Yankton; Northern Hospital for the Insane, Redfield.)

The hospitals are each in charge of a superintendent, holding office during the pleasure of the State Board of Charities, appointed by the governor with the consent of the senate. The superintendent is bonded for the faithful performance of his duties, and is the chief executive officer of the hospital. (Annotated Statutes of South Dakota, §§ 430, 439.)

Residents of the State receive treatment free. Residents of other States may be admitted, when convenient, and pay for their treatment. Patients may arrange to have special care and treatment. (Idem, § 44.)

Whenever a patient becomes entirely cured, he is discharged by the superintendent. Patients not cured may be discharged upon arrangement with the Commissioners of Insanity of the county of residence, suitable provisions being made for their care. (Idem, §§ 448, 449.)

In each county there is a board of commissioners of insanity, the chairman of which is the county probate judge, the other two members being a physician and attorney, respectively. They have cognizance of all applications for committment within their respective counties. They have the powers of a court as to demanding the presence of witnesses, etc. (Idem, §§ 2972, 2975.)

Applications for admission to the hospital shall be made in writing in the nature of an information verified by affidavit, showing the fact of insanity and the residence of the patient. On the filing of the information the commission investigates the case, may command the presence of witnesses, and shall appoint a physician to make a personal examination of the patient and report. (Idem, §§ 2976–2978.)

On the physician's certificate the commission concludes the investigation and determines the question of insanity. If they find insanity, they certify the fact. The certificate is handed to the sheriff, who shall deliver the patient, together with the certificate, to the superintendent of the hospital. (Idem, § 2978.)

Whenever the legality of the detention is questioned, the county judge may appoint a commission to investigate and report to him. Upon the return of this report the county judge may order the patient's discharge. (Idem, §§ 2986, 2987.)

The expense of care and treatment falls on the county of the patient's residence. (Idem, § 2996.)

Patients have leave to correspond without restriction with one person whom the patient may select. (Idem, § 3006.)

TENNESSEE.

(Central Hospital for the Insane, Nashville; Eastern Hospital for the Insane, Knoxville; Western Hospital for the Insane, Bolivar.)

Jurisdiction.—Jurisdiction over the persons and estates of idiots, lunatics, and other persons of unsound mind is vested in the county and chancery courts. (Code of Tennessee, 1896, § 5451.)

Inquisition.—Upon information made to the County Court that any idiot or lunatic resides within the jurisdiction thereof the court shall order the sheriff to summon a jury of twelve freeholders to ascertain by inquisition the idiocy or lunacy, and the property and estate of the idiot or lunatic, and make return thereof to the court. Witnesses may be subpenaed and are subject to the penalties and entitled to the privileges of other witnesses. If the person is not declared a lunatic, the person on whose application the inquisition is issued is liable for costs. Upon the return of the jury that the person is an idiot or lunatic, and that he has property, the court shall appoint a guardian for the person and property of such idiot. If the idiot or lunatic has no property, or not sufficient for his maintenance, he may be let out for the term of one year to the lowest bidder as other poor persons, or be otherwise provided for as the court may direct. If let out to the lowest bidder, bond and sufficient security as prescribed by the court shall be taken for the safekeeping, providing sufficient diet, washing, and apparel, and proper treatment for the term of letting. (Idem, §§ 5453, 5460.)

Inquisition in the Chancery Court.—The application to the Chancery Court shall be by petition verified by affidavit setting forth the facts in regard to the person and property of the supposed idiot or lunatic. No application shall be made unless the value of the property exceeds five

hundred dollars. The chancellor shall direct the issuance of a writ of inquisition upon the giving of a bond by the petitioner conditioned to pay costs and all such damages as the defendant may sustain in consequence of the petitioner having wantonly and maliciously instituted proceedings. The jury consists of twelve freeholders, and they are required to ascertain by their verdict whether the defendant be an idiot, lunatic, or person of unsound mind. Notice of the time and place of the inquest shall be given to the alleged lunatic at least five days previous to the time of hearing; if the jury finds the person to be a lunatic, the verdict shall ascertain the value of the estate and of what it consists, and who are the next of kin of the insane person. Upon such finding the clerk shall appoint a guardian to take care of the estate and person of the insane person. Upon motion after the return of the inquisition the verdict of the jury may be set aside, and the chancellor may thereupon order another inquest to be held. If the jury disagree, the chancellor may in his discretion order another inquest or decide the case himself upon the testimony returned and such other testimony as may be offered. (Idem, §§ 5461, 5477.)

Powers of Guardian.—A guardian may, upon the coming of age or marriage of the child of any confirmed lunatic, make such settlement upon such child as the value of the estate, the condition of the lunatic and his wife, and other circumstances may render reasonable and just. The guardian may apply in such case to the chancellor for direction. The real and personal property of a person laboring under confirmed mental unsoundness may be portioned by the court among his children or descendants as in case of death and attestacy, such portion to be charged as an advancement. The property of a person of unsound mind may be sold upon the petition of the guardian if it appear manifestly for the interest of such person, and the proceeds shall be disposed of by the court in such manner as best to promote the interests of the owner. Guardians of lunatics, idiots, and other persons of unsound mind shall be punishable for the same abuses, mismanagements, neglects, failures, and other offences as guardians of minors and in the same manner. (Idem, §§ 5478–5490.)

County Asylums.—Any county of the State having a population in 1880 of over 40,000 may erect and maintain an asylum for the insane, the poor, afflicted, and inebriates. Managing and building commissioners are appointed by the county judge, three of whom shall be justices of the peace and three citizens and residents of the county. Applications for admission are made to the commissioners, who examine and admit or reject the applicant. (Idem, §§ 2733, 2734.)

Hospital for the Insane.—The Tennessee hospitals for the insane are each governed by a board of fifteen trustees. The board shall appoint a superintendent to the hospital who shall be a skilful physician of unblemished moral character, of enlightened and thorough professional education, of prompt business habits, and of humane and kind disposition. He shall be a married man, and with his family shall reside constantly in the institution. It shall be his duty to exercise a general supervision over all matters relating to the hospital, to visit the patients therein at least twice a week, to call special meetings of the board whenever it may be deemed necessary, to report to the trustees annually the number of patients admitted in the hospital, the date of admission of each patient, the degree and kind of insanity with which each patient is afflicted, the length of time supposed to have been afflicted before admission, the previous occupation, age, and habits of each patient, the names and addresses of those discharged and the condi-

tion of each one discharged, and such other particulars as he may deem necessary. (Idem, §§ 2578–2605.)

Admission of Patients.—Insane persons may be placed in the hospital by their legal guardians, or by their relatives or friends in case they have no guardian, or by a justice of the peace if the person be proved to be insane. Non-paying patients may be admitted to the number of one to every eighteen hundred of the population of each county, upon a statement in writing of some reputable citizen of the county to the effect that the person is insane, and that his insanity is of less than two years' duration, that he is in needy circumstances and is a citizen of the State of Tennessee. The justice shall issue subpœnas for persons named as witnesses and such other persons as he may think proper. If after such inquest the justice is satisfied of the truth of the allegations contained in the statement, he shall require the medical witnesses to certify that they have examined the person alleged to be insane and as to his condition. If satisfied that the person alleged to be insane is insane, the justice shall certify to such fact and transmit to the clerk of the County Court a certificate of the proceedings had. The clerk shall file such certificate in his office and transmit a copy of the same to the superintendent of the hospital, accompanied by an application for the admission of the patient therein named. Upon the receipt of the application the superintendent shall notify the clerk as to when the patient can be received. (Idem, §§ 2613–2618.)

If a person admitted to a hospital for the insane have estates the income of which is sufficient to pay a part but not all the cost of his maintenance in the hospital, such income may be applied in payment thereof if such insane person has no family dependent upon him. (Laws of Tenn. of 1899, ch. 428.)

TEXAS.

(Hospital for the Insane, Austin; North Texas Hospital for the Insane, Terrell; Southwestern Insane Asylum, San Antonio.)

Persons of unsound mind are idiots, lunatics, or insane persons. An habitual drunkard is one whose mind has become so impaired by the use of intoxicating liquors or drugs that he is incapable of taking care of himself or property. (Revised Statutes of Texas, arts. 2533 and 2554.)

Jurisdiction.—The County Court has power to appoint guardians of persons of unsound mind and habitual drunkards, settle the accounts of such guardians, and transact all business pertaining to the estates of such persons. The District Court has appellate jurisdiction over the County Court in all matters of guardianship, and original jurisdiction under such regulations as may be prescribed by law. (Idem, arts. 2550 and 2551.)

Appointment of Guardians.—Proceedings for the appointment of guardian of the persons, estate, or of either, of persons of unsound mind or habitual drunkards, shall be commenced in the county where such person of unsound mind or habitual drunkard resides. The nearest of kin of such person who is not disqualified shall be entitled to the guardianship, and where two or more are equally entitled the guardianship shall be given to the one or the other according to circumstances, taking into consideration the interests of the ward alone. If such ward have a husband or wife who is not disqualified, such husband or wife shall be entitled to the guardianship in preference to any other person. Notice of the application for the appointment of a guardian shall be given to the alleged person of unsound mind or habitual drunkard.

Before appointing a guardian the court shall be satisfied that the person for whom the guardian is to be appointed is a person of unsound mind or an habitual drunkard, that the court has jurisdiction in the case, and that the person to be appointed guardian is not disqualified to act. (Idem., arts. 2566, 2583, 2587, 2588.)

Guardians of persons of unsound mind and habitual drunkards are required to take oath and give a bond, and their powers and duties with regard to the estate of their wards are the same as those of guardians of minors. (Idem, title 51, ch. 6.)

Proceedings for the Appointment of Guardians.—Upon information given to the judge of a County Court that any person is of unsound mind or is an habitual drunkard and is without a guardian, the judge, if satisfied that there is good cause, shall issue a warrant to the proper officer commanding such person to be brought before him at a time and place to be named in such warrant. The information shall be in writing, stating the name of the person charged, and that such person is of unsound mind or is an habitual drunkard; such information shall be subscribed and sworn to by the informant. A jury shall be impanelled to try the case and decide whether such person is of unsound mind or is an habitual drunkard. The case shall be conducted in the name of the county as plaintiff, and the person against whom the information is filed as defendant, and the proceedings and trial therein shall be governed in the same way as in ordinary suits in the County Court unless otherwise provided. If it be found that the defendant is of unsound mind or an habitual drunkard, the court shall immediately appoint a guardian of the person and estate of such defendant. (Idem, arts. 2735–2740.)

When Ward is Furiously Mad.—If any person shall be furiously mad, or so far disordered in his mind as to endanger his own person or the person or property of others, it shall be the duty of the guardian to confine him in some suitable place until the first regular term of the County Court, when the court shall make such order for the restraint, support, and safekeeping of such ward as circumstances may require. If any such person is not so confined, any magistrate may cause him to be apprehended, and employ any person to confine him in some suitable place until the County Court shall make further order thereon. (Idem, arts. 2745, 2746.)

Maintenance of Insane Persons.—Where a person of unsound mind or an habitual drunkard has no estate of his own, he shall be maintained: 1. By the husband or wife, if any, if able to do so. 2. By the father or mother, if able to do so. 3. By the children and grandchildren, if able to do so. 4. By the county in which such person has his residence. The expense attending the confinement shall be paid by the guardian out of the estate of the ward, if he has any, and if not, by the person bound to provide for him; if not so paid, the county shall be chargeable with such expense. (Idem, arts. 2747, 2748.)

Lunatic Asylums.—The control of each Texas asylum for the insane is vested in a board of managers, consisting of five members appointed by the governor. They shall have original jurisdiction and control of all the property and business of the asylums. They may make by-laws and regulations for the government of the institutions, determine the salary and wages of officers, discharge officers, employés, or patients, appoint assistant physicians, suitable matrons, and apothecary, examine the accounts and vouchers of the superintendent, exercise a careful supervision over the general operations and expenditures of the asylums, and direct the manner in which their

revenue shall be disbursed. The committee shall visit each asylum once every month, and the whole number once a year. (Idem, arts. 87–96.)

Powers of the Superintendent.—The superintendent shall be elected by the board of managers of the asylum. He shall be a married man, a skilful physician, and experienced in the treatment of insanity. He shall reside at the asylum, and devote his whole time to the duties of his office. He shall be the chief executive and medical and disbursing officer of the institution, and, subject to the by-laws, shall have general care and control over everything connected therewith. With the consent of the board of managers he shall employ such officers and other persons as may be required. He shall keep a register of all patients received into the asylum and discharged therefrom. (Idem, arts. 97–105.)

Admission of Patients into Asylum.—All persons adjudged insane and ordered confined by a court of competent jurisdiction may be admitted into an asylum. This class shall be known as public patients. All persons certified to be insane by some reputable physician under the regulations prescribed by statute shall be received, and shall be known as private patients. Before any person can be received as a private patient the guardian, or parent, or some near relative or other person interested in him, shall present a written request to the superintendent for his admission, setting forth such facts as may be required by the superintendent, which written request shall be under oath of the party presenting it, and be accompanied with the affidavit of the physician certifying to the insanity. Such application shall also be accompanied by a certificate of the county judge that the physician certifying to the insanity of the person is a reputable physician in regular practice. All private patients shall be kept and maintained at the asylum at their own expense or at the expense of their relatives or others. All indigent public patients shall be kept and maintained at the expense of the State. (Idem, arts. 112–115.)

Judicial Proceedings in the Case of Lunacy.—Upon information in writing and under oath to the county judge that any person in his jurisdiction is a lunatic, and that the welfare of himself or of others requires that he be placed under restraint, and if such county judge shall believe such information to be true, he shall issue his warrant for the arrest of such person and fix a day for a hearing. A justice of the peace may issue the warrant for such apprehension, to be returnable to the County Court. A jury of twelve competent persons shall be summoned and duly examined and sworn. Evidence shall be produced and heard, and the county judge shall submit to the jury the question as to whether the defendant is of unsound mind, and whether he should be placed under restraint. If this question be decided in the affirmative, the verdict of the jury shall state the age and nativity of the defendant, the number of attacks of insanity he has had, and whether or not insanity is hereditary in the family of defendant. Judgment shall thereupon be entered adjudging the defendant to be a lunatic and ordering him to be committed to the lunatic asylum for restraint and treatment. (Idem, arts. 128–135 as amended by Laws of 1900, ch. 106.)

UTAH.

(Utah State Insane Asylum, Provo City.)

The State Insane Asylum of Utah is located at Provo City, Utah County. Its government and control are vested in a board of commissioners, consisting of the governor, State treasurer, and State auditor, known as the Board

of Insane Asylum Commissioners. (Revised Statutes of Utah, §§ 2153, 2155.)

Powers.—The board may contract and be contracted with, and may sue and be sued, in all matters concerning the asylum. It may take, hold, purchase, give, or devise real or personal property required for its uses (§ 2151). It may make by-laws for its government and that of the asylum (§ 2158), and holds meetings at the asylum monthly, and keeps a record of its proceedings open at all times to the public (§ 2158). The board has control of all insane persons in the State, and may remove any employé of the asylum (§ 2161). It shall certify in October of each year to the State auditor the number of patients in the asylum classified as indigent and paying (§ 2162).

The board has a secretary and a treasurer, who have the usual duties and responsibilities. (Idem, §§ 2162, 2163.)

Medical Superintendent.—The board appoints a medical superintendent, who resides at the asylum and holds his office during the pleasure of the board. He shall be a graduated physician of five years' standing and actual practice. He is the chief executive officer of the asylum and has general superintendence. He controls the care of the patients and prescribes the method of treatment. He shall keep a record of all proceedings and makes reports quarterly as to the condition and needs of the asylum. The board examines his reports and submits the same with their recommendations to the State auditor. (Idem, § 2165.)

The superintendent shall keep a full record of the proceedings day by day, and shall report as to expenses, etc., monthly, with vouchers, to the board. He shall visit the wards of the asylum every day unless excused. (Idem, § 2168.)

An assistant physician may be provided in the discretion of the board. There is also a steward. (Idem, § 2166.)

Commitment.—The judges of the District Courts have cognizance of all applications for admission to the asylum or for the safekeeping of insane persons within their respective districts. In the absence of such a judge, the judge of another district may act, or the chairman of the Board of County Commissioners may commit temporarily until the return of the judge (§ 2170). The application for admission to the asylum shall be made in the form of an information duly verified, alleging that the person is believed to be insane, and the fact of residence in the county (§ 2173). On the filing of such information the judge of the district for the county of residence may examine the informant and may have the alleged incompetent brought before him (§ 2174). Testimony for and against the application shall be heard. The judge shall call two physicians, before whom he shall examine the charge (§ 2175). After the hearing and examination the physicians shall certify as to the insanity or otherwise of the alleged incompetent and whether dangerous (§ 2176). On the filing of the physicians' certificate the judge concludes the investigation, and if he find the person insane, orders his commitment (§ 2177). The sheriff executes the order and takes the person committed to the asylum, together with a certified copy of the papers in the case, whereupon the superintendent receives the patient (§ 2180). If the patient has property, a guardian is appointed by the committing judge (§ 2181). The board may order the discharge of a patient who proves to be harmless or incurable, and order him cared for by the county commissioners of the county of residence (§ 2186).

No non-resident may be cared for in the State Asylum, but shall be

ordered discharged when discovered to be an inmate, and returned. (Idem, § 2187.)

Friends, through the judge committing, may arrange for the care of a patient outside the asylum by furnishing a bond. (Idem, § 2188.)

Upon an affidavit as to lack of insanity furnished the board, the board shall investigate, and if it finds the patient to be wrongfully detained, shall immediately order his discharge. (Idem, § 2190.)

The inmate's estate is liable for his care until exhausted. (Idem, § 2192.)

An inmate's friends or relatives may arrange, by paying therefor, for special care and treatment for the patient. (Idem, § 2193.)

VERMONT.

(Vermont State Hospital for the Insane, Waterbury.)

The words "insane person" shall include every idiot, *non compos*, lunatic, and distracted person. (Statutes of Vermont, § 7.)

The word "spendthrift" includes every person who is liable to be put under guardianship on account of excessive drinking, gaming, idleness, or debauchery. (Idem, § 2750.)

Appointment of Guardian.—The Probate Court may appoint guardians of insane persons or spendthrifts on the application of a relative or friend, or of the overseer of the poor of the town in which the person resides or is chargeable, representing to the court that such person is insane and incapable of taking care of himself and praying that a guardian be appointed. The court shall fix a time for considering the application, notice of which shall be given to the alleged insane person or spendthrift at least twelve days before the time set. The court shall investigate the case and make such decree in the premises as appears just. If a guardian is appointed, the costs of the ward in defending against the application shall be paid out of the ward's estate. (Idem, §§ 2751-2762.)

Powers of Guardians.—Guardians shall, until they are legally discharged, have the possession and management of the estates of their wards and the care and custody of such members of the families of their wards as are dependent upon them for support, education, or employment, unless they have other guardians. (Idem, § 2760.)

The general provisions applicable to the care and management of the estates of minors are also made applicable to the guardians of insane persons and spendthrifts. The estates of such wards are controlled subject to the direction of the Probate Court, and no disposition thereof can be made except upon the order of such court. (Idem, § 2735.)

Insane Poor.—Insane persons in any town destitute of the means to support themselves and having no relatives bound by law to support them, and having no legal settlement in any town, shall be supported by the State at the insane asylum. (Idem, § 3213.)

The selectmen of a town shall ascertain whether an insane person is liable to be supported by the State, and for such purpose may institute a court of inquiry before the judge of probate. If the probate judge finds that such person should be supported by the State, and his insanity is certified by two legally qualified physicians residents of the State, he shall issue an order for the removal of such person to the State Asylum at Waterbury or the Brattleboro retreat at Brattleboro. (Idem, §§ 3214-3222.)

Supervision of the Insane.—The governor shall appoint three super-

visors of the insane, who shall hold their offices for six years. Two of such supervisors shall be physicians, and none of them shall be a trustee, superintendent, employé, or other officer of an insane asylum in the State. The supervisors shall visit the Vermont State Hospital for the Insane and the Brattleboro Retreat as often as occasion requires, and one member as often as once a month, and also any other place where insane persons are confined in the State, at their discretion. They shall examine into the condition of the asylums in such other places, the management and treatment of the patients therein, their physical and mental conditions and medical treatment; form a careful opinion of the patients, apart from the officers and keepers, and investigate the cases that in their judgment require special investigation, and particularly ascertain whether persons are confined in such asylums or other places who ought to be discharged. (Idem, §§ 3227–3229. By Laws of 1898, No. 63, the name of the Vermont State Asylum was changed to the Vermont State Hospital for the Insane, and the word "asylum" or "asylums" wherever appearing in Chapters 146, 147, and 148 of the Statutes of Vermont, was changed to "hospital" or "hospitals.")

Admission to Insane Asylum.—No person shall be admitted to or detained in the insane asylum as a patient or inmate except upon the certificate of such person's insanity, made by two physicians of unquestioned integrity and skill, residing in the probate district in which such insane person resides. Such certificate shall be made not more than ten days previous to the admission of such insane person. Such certificate shall be verified before a magistrate, who shall append thereto his jurat, and certify therein that such physicians are of unquestionable integrity and skill, and shall be presented to the proper officer of the asylum at the time such insane person is presented for admission. The certificate of the physicians shall be given only after a careful examination of the patient, made not more than five days previous to making the certificate. A person may be received into an asylum without a certificate upon the order of the Supreme or County Court upon the presentation of a certified copy of the order or sentence. A person admitted to an asylum in pursuance of law shall be deemed insane, and be subject to the control and sanitary treatment of the trustees of the asylum, until sufficiently sane to warrant his release, or until removed by his friends or guardians or otherwise discharged. (Idem, §§ 3239–3249.)

Idiots not to be Admitted.—Idiots and persons *non compos*, or demented persons who are not dangerous, shall not be confined in a hospital or retreat for the insane. The supervisors of the insane shall discharge all such persons so confined, and the superintendent of the hospital or retreat, or the board of trustees, shall notify the selectmen or mayor of the town or city from which such person was sent of such discharge. If such person is not removed by such selectmen or mayor within twelve days after such notice, the support of such person becomes a charge on such town or city. (Idem, § 3245, as amended by Laws of 1898, Act No. 63.)

VIRGINIA.

(Eastern State Hospital, Williamsburg; Central State Hospital, Petersburg; Western State Hospital, Staunton; Southwestern State Hospital, Marion.)

The word "insane person" shall be construed to include every one who is an idiot, lunatic, *non compos*, or deranged.

Appointment of Committees of Insane Persons.—If a person residing in this State be thought insane, the court of a county or corporation of

which such person is an inhabitant shall, on the application of any party interested, proceed to examine into the state of mind of the alleged insane person, and, being satisfied that he is insane, appoint a committee of him. The Circuit Courts have jurisdiction with the County and Corporation Courts in the appointment of committees. The committee is required to give a bond in such penalty as the court may deem sufficient. If the person appointed committee refuses the trust, or fails within two months from the date of his appointment to give a bond as required, the court, on the motion of any party interested, may appoint some other person a committee. (Code of Virginia, §§ 1698–1701.)

Powers and Duties of Committees.—The committee is entitled to the custody and control of the person, and shall take possession of the estate of the insane person. He may sue and be sued in respect thereto, and for the recovery of debts due to or from the insane person. He shall preserve such estate and manage it to the best advantage. He shall apply the personal estate, or so much as may be necessary, to the payment of the debts of such insane person, and the rents and profits of the residue of his estate and real and personal, or so much as may be necessary, to the maintenance of such insane person and of his family. If the personal estate be insufficient for the support and maintenance of the insane person, the committee may petition the court for the sale of the real estate. Upon presentation of such petition, it shall be referred to a commissioner in chancery, who shall inquire into and hear all parties interested in such estate, and report thereon with all convenient speed. If it shall appear to the court proper, an order shall be entered for the mortgaging, leasing, or sale of so much of the real estate as may be necessary. (Idem, §§ 1702–1705.)

State Hospitals.—The state hospitals are under the management of boards of directors, consisting of nine members, whose term of office is three years. Each board appoints a superintendent of each hospital, who shall be a physician, and may appoint an executive committee, and such officers, nurses, and attendants as they may deem proper, and prescribe their compensation. (Idem, §§ 1660, 1661, as amended by Acts of 1893, 1894, p. 397, 1662–1665.)

Commitment to Hospitals.—Any justice suspecting any person in his county or corporation to be a lunatic shall issue his warrant ordering such person to be brought before him. He and two other justices shall inquire whether such person be a lunatic, and for that purpose summon a physician and any other witnesses. If the justices decide that the person is a lunatic and ought to be confined in an asylum, they shall order him to be removed to the nearest asylum and received, if there be room therein, and if not, to either of the others, unless some person will give a bond for the proper care of such lunatic without cost to the Commonwealth. The questions asked of the witnesses and the answers thereto shall be in writing, and, together with a written statement by the justices of any matter known to them as to the effect of insanity, shall be transmitted by them with the order. If there be no vacancy in the asylum, the patient shall be kept in the jail of the county or corporation. When such patient arrives at the asylum the superintendent and his assistants shall examine him, and if they concur with the justices, shall receive and register him as a patient. (Idem, §§ 1669–1675.)

Expenses of Maintenance of Insane.—If the insane person be possessed of property, all the expense of his removal to and from the asylum, and his maintenance and care therein, shall be paid out of such property. The directors of either asylum on behalf of such asylum, and the auditor of

public accounts on behalf of the Commonwealth, may release such person of the payment of such expense if he have a family dependent upon his estate for support, or in their opinion it be just and equitable that such claim should be so released. (Idem, §§ 1706–1708.)

WASHINGTON.

(Western Washington Hospital for the Insane, Fort Steilacoom; Eastern Washington Hospital for the Insane, Medical Lake.)

Guardians of Persons of Unsound Mind.—The Superior Courts have power to appoint guardians to take the care, custody, and management of idiots, insane persons, and all who are incapable of conducting their own affairs, and of their estates real and personal, the maintenance of themselves and families, and the education of their children. If it be found by the court that the person so brought before the court is of unsound mind and incapable of managing his affairs, the court shall appoint a guardian for the estate of such insane person. If the person alleged to be insane shall be discharged, the court, in its discretion, may order the costs to be paid by the person at whose instance the proceeding was had. Each guardian, before entering upon the duties assigned to him, shall enter into a bond to the board of county commissioners in such sum and with such security as the court shall approve. Every such guardian shall give notice of his appointment within twenty days thereafter. (Ballinger's Codes and Statutes, 1897, vol. ii., §§ 6424–6429.)

Powers and Duties of Guardians.—Such guardian shall collect and take into his possession the goods, chattels, moneys, effects, and other evidences of debt, and all writings touching upon the estate real and personal of the person under guardianship. Within forty days after his appointment he shall make an inventory of the real and personal estate of his ward. Such inventory shall be attested and verified by the oath of his guardian. He shall prosecute all actions commenced by or on account of his ward, and defend all actions brought against such ward. He shall collect all debts due his ward, and pay all demands due and become due from his ward. (Idem, §§ 6429–6433.)

Power of Court as to Insane.—The Superior Court shall have power to make orders for the restraint, support, and safekeeping of such person, for the management of his estate, the support and maintenance of his family, and the education of his children, out of the estate; to set apart and reserve for the use of such family all property real or personal not necessary to be sold for the payment of debts; and to let, sell, or mortgage any part of such estate real or personal not necessary for the payment of debts, the maintenance of such insane person or his family, or the education of his children. (Idem, § 6434.)

Commitment of the Insane to Hospitals.—The judge of the Superior Court of any county, upon the application of any person under oath, setting forth that any person by reason of insanity is unsafe to be at large, shall cause such person to be brought before him, and summon to appear two or more witnesses, who shall testify as to the conversation, morals, and general conduct upon which such charge of insanity is based, and also two reputable physicians before whom a judge shall examine the charge, unless a jury is demanded, to decide upon the question of insanity. If a jury be demanded, the trial shall be by jury. The physicians, after a careful hearing of the case and a personal examination of the alleged insane person,

shall certify as to the insanity of such person. If such person be declared insane, the court shall order him to be committed to a hospital for the insane. He shall issue a warrant of commitment directed to the sheriff, demanding him to convey such insane person to the hospital. He shall transmit a copy of the complaint and commitment and physician's certificate, which shall always be in the form as furnished to the courts by the superintendent of the hospital. The costs of the commitment are a county charge. No case of idiocy, imbecility, or harmless chronic mental unsoundness shall be committed to a hospital for the insane; and whenever, in the opinion of the superintendent, after a careful examination of the case of any person committed, it shall be satisfactorily ascertained by him that the party has been unlawfully committed, and that he or she comes under the rule of exceptions provided for herein, he shall have authority to discharge such person and return him to the county from which he was committed. (Idem, §§ 2660–2663, 2666.)

Government of Hospitals for the Insane.—A State Board of Audit and Control has charge of the State hospitals, together with the Reform School, the Soldiers' Home, and the State Penitentiary. Such board is composed of five members appointed by the governor, by and with the consent of the senate, four of whom serve without compensation, and the fifth member is paid a salary, and is known as the Commissioner of Public Buildings. Such board has power to make all repairs and improvements which in its judgment may be necessary for the conduct of the hospitals under its charge. It shall take charge of the general interests of the hospitals, and manage and conduct the same in such manner as may appear to it best and most economical. It shall appoint a superintendent and make by-laws for the government of each hospital; and prescribe in a manner consistent with the laws of the State the duties of all persons connected in any way with the management of the hospitals under their charge. The superintendent shall be a skilful physician and shall reside in the hospital. He shall hold his office for such time as the trustees may deem wise and for the efficiency and economy of the institution; he shall have entire control of the medical, moral, and dietetic treatment of the patients, and, so far as is not inconsistent with the by-laws and regulations of the hospital, of all other internal government and economy of the institution. (Idem, §§ 2621–2629, 2655–2659.)

WEST VIRGINIA.

(West Virginia Hospital for the Insane at Weston, Weston; West Virginia Asylum for Incurables at Huntington, Huntington; West Virginia Hospital for the Insane at Spencer, Spencer.)

Jurisdiction Over Insane.—The County Court has original jurisdiction in all matters pertaining to the appointment of committees and curators for insane persons in the settlement of their accounts. (Code of West Virginia, 1899, ch. 39, § 9.)

By the constitution, art. 8, § 24, the County Court is given original jurisdiction in the appointment of committees of insane persons in the settlement of their accounts, but the jurisdiction of the County Court by that section is made subject to such limitations as may be prescribed by law.

Appointment of Committees.—If a person be found insane by the justices before whom he is examined, the Circuit Court of the county of which he is a resident shall appoint a committee of him. If the person not so found be suspected of insanity on the application of any party inter-

ested, the Circuit Court shall proceed to examine into his state of mind, and being satisfied that he is insane, appoint a committee. The committee is required to give a bond, approved by the court making the appointment, for the faithful performance of his duties. (Idem, ch. 58, §§ 33, 34, 36.)

Powers and Duties of Committee.—The committee of an insane person has the custody and control of his person and possession of his estate. He shall take care of and preserve his estate, and manage it to the best advantage; shall apply the personal estate, or so much as may be necessary, to the payment of the debts of such insane person, and the rents and profits of the residue of his estate real and personal, and the residue of his personal estate, to the maintenance of such insane person and of his family. The real estate may be sold, if the personal estate be insufficient for the payment of his debts and for the support and maintenance of himself and his family, upon petition to the court by which the committee was appointed for authority to make such sale. (Idem, ch. 58, §§ 37–41.)

Commitment of Lunatics.—Any justice suspecting any person in his county to be a lunatic, shall issue his warrant ordering such person to be brought before him. He shall inquire whether such person be a lunatic, and for that purpose summon a physician and other witnesses. He may propound such questions as he may deem fit, including those prescribed by statute. If the justice decides that the person is a lunatic and ought to be confined in a hospital, the said justice shall order him to be removed to the hospital, and to be received there if there is room therein, unless some person to whom the justice in his discretion may deliver such lunatic will give bond with condition to restrain and take proper care of such lunatic until the cause of confinement shall cease. The questions and answers of the witnesses shall be in writing, and together with a written statement by the justice of any matter known to him as to the fact of insanity, shall be transmitted by him with the order. If there is a vacancy in the hospital, the sheriff shall convey the lunatic to the hospital; and if the examining board concur in the opinion of the justice as to the person's insanity, he shall be received and registered as a patient. If they refuse to receive him, he shall be confined in the jail of the county in which he was examined until lawfully discharged or removed therefrom.

State Hospitals for the Insane.—The state hospitals for the insane are each under the management of a board of directors composed of nine members appointed by the governor. They have charge of the hospital, and of any real or personal property conveyed to it for its use. They shall employ a superintendent, who shall receive such compensation as the board may prescribe, and may also appoint such officers, nurses, and attendants as they may see fit. Any one or more of the directors, together with the superintendent, constitute an examining board, and may examine persons brought to the asylum as lunatics, and order those found to be such to be received, and those found to be restored to be discharged therefrom. (Idem, ch. 58, §§ 1–8.)

WISCONSIN.

(Wisconsin State Hospital for the Insane, Mendota; Asylum for the Chronic Insane, Wauwatosa; Northern Hospital for the Insane, Winnebago; Milwaukee Hospital for the Insane, Wauwatosa.)

Guardians for Insane.—Whenever it shall be represented to the County Court by a verified petition of any relative or friend of any insane person that any person by extreme old age or other cause is mentally incompetent

to have the charge and management of his property, said court shall cause a notice to be given to the supposed insane or incompetent person of the time and place of hearing the case, not less than twenty days before the time appointed, and shall cause such person, if able to attend, to be produced before him on the hearing. If such person is confined in a State or county hospital for the insane, the notice may be served on the superintendent thereof; and if in the opinion of such superintendent it is not proper to remove such insane person, such fact shall be certified to the court, and it shall be deemed a sufficient cause for not producing such insane person on the hearing. If, after a full hearing and examination, it shall appear to the County Court that the person in question is incapable of taking care of himself and managing his property, the court shall appoint a guardian of his person and estate. (Statutes of Wisconsin, 1898, §§ 3976, 3977.)

Guardian for Drunkards.—When any person, by excessive drinking, gaming, idleness, or debauchery of any kind, shall be unable to attend to business, and shall thereby greatly endanger his health, life, or property, or so spend, waste, or lessen his estate as to endanger his own or his family's support, any friend or relative, or any supervisor or justice of the peace, may present a verified petition to the County Court praying to have a guardian appointed. Notice shall be given to such person not less than twenty days before the time fixed for the hearing. The court shall appoint a guardian of his person and estate if after a full hearing it shall appear proper. The court may authorize a guardian of any person to commit such person to any inebriate asylum. (Idem, § 3978.)

Powers of Guardians.—Guardian shall collect and pay the debts of his ward, and manage his estate frugally and without waste, and apply the income and profits thereof for the suitable maintenance and support of the ward and his family, if there be any. If such income and profit shall be insufficient for that purpose, the guardian may sell the real estate upon obtaining a license therefor as provided by law, and shall apply the proceeds of such sale, so far as may be necessary, for such maintenance and support. (Idem, §§ 3982, 3983.)

Proceedings to Determine Insanity.—Upon the application of any respectable citizen to the judge of the County Court, judge of the Circuit Court, or any judge of a court of record, or any judge or justice of a police court of a city of the first class, a judicial inquiry may be had as to the mental condition of the person supposed to be insane. The application shall be in writing, and shall specify whether or not a trial by jury is desired by the application. On receipt of the petition the judge shall appoint two disinterested physicians of good repute, of medical skill and moral integrity, to visit and examine the person alleged to be insane; and such physicians shall proceed to the residence of the person supposed to be insane and shall, by personal examination and inquiry, satisfy themselves as to his condition, and report the result of their examination to the judge. The form of such report is prescribed by statute. Upon receiving the report of the examining physicians the judge may, if no demand has been made for a jury, make and enter his order of commitment to the hospital or asylum of the district to which the county belongs; or if not fully satisfied, he may make such additional investigation of the case as may seem to him necessary and proper. In case a trial by jury is demanded, the forms of procedure shall be the same as in trials by jury in justices' courts, and the trial shall be in the presence of the person supposed to be insane and his counsel and immediate friends and the medical witnesses. If the jury finds the supposed person to

be insane, their verdict shall simply so state. If the jury by their verdict finds the supposed insane person is sane, the judge shall enter an order for the discharge of such person. (Idem, § 593, 1898, as amended by Laws of 1899, ch. 21.)

Inquiry as to the Sanity of the Person Confined.—Application may be had in the behalf of any person confined in any hospital, asylum, or other place of confinement, by any reputable citizen having reason to question the propriety or justice of such confinement, to a judge of the County Court of the county in which such person is confined, or to a judge of the County Court by whom he was so committed. The judge shall have and may exercise the authority and jurisdiction in the matter of such application as is conferred upon him by Section 593 as to the original commitment, and the same proceedings may be had in such matter. If, upon such rehearing and further inquiry, it shall be found that the person so confined is sane, the judge shall make an order for the release of such person. (Idem, § 593a.)

Admission of Patients to State Hospital.—Patients shall be admitted from the several counties to the State hospitals in ratio of population. No county shall at any time have more than its just proportion of patients in a hospital unless some other county has less than its proportion. If an inmate in a State hospital is improperly charged to the State or to any county, the attorney-general on behalf of the State, and the district attorney of such county on its behalf, may make written application to the Board of Trustees of State Hospitals for relief from the charge. A hearing shall be had upon such application. Such application may be supported by affidavits and other proper evidence. If, upon such hearing, the board is satisfied that the relief asked for should be granted, it shall, by its order in writing, grant the same and name the county chargeable with such inmate. (Idem, § 583.)

Hospital Districts.—The State is divided into two hospital districts. All patients in the counties in each hospital district shall be sent to the hospital therein situated. (Idem, § 581.)

Superintendent of Hospital.—The superintendent of each hospital shall be the chief executive officer of the hospital, and devote all his time and attention to his duties. He shall exercise entire control over all the subordinate officers. He shall hire all employés and assistants necessarily connected with the institution below the grade designated in the by-laws as officers, and may discharge any officer, assistant, or employé at will, being responsible to the board for the proper exercise of that duty in regard to officers. (Idem, § 582.)

WYOMING.

(State Hospital for the Insane, Evanston.)

The territorial treasurer is custodian of the funds allowed for the support of the state hospital. The hospital is under the direct supervision of a Board of Commissioners, who keep the accounts and records of the hospital and of their proceedings and report to the legislature. (Revised Statutes of Wyoming §§ 2757, 3762.)

Superintendent; Qualifications and Duties.—The Board of Commissioners elects one resident physician to be general superintendent of the hospital. He shall be a graduated physician, reside at the hospital, and have the care thereof and the control of its methods of treatment, etc. He shall

keep a record of the matters under his control, and shall report monthly to the Board of Commissioners. (Idem, § 3764.)

Commissioners in each county commit patients in their respective jurisdictions to the hospital. (Idem, § 3765.)

Paying patients may be admitted upon terms approved by the Board of Commissioners. (Idem, § 3766.)

Commitment.—Commitment is upon information in writing to the probate judge of the county of residence, and upon his finding cause therefor the question of insanity is referred to a jury. The court may require the patient's presence in court on the hearing. Upon the determination of the jury certificate is made; and if insanity is found, the probate judge orders the commitment. Guardians of incompetent persons are appointed by the probate judge. If a person becomes violent, it is the duty of the guardian properly to keep and protect the insane person until such a time as the probate judge may order his restraint or commitment. (Idem, §§ 2287, 2329.)

THE POWERS AND DUTIES OF COMMITTEES OF INCOMPETENT PERSONS.

The insane are deemed by the law incapable of managing their own affairs. When a person becomes mentally disabled, from whatever cause the disability may arise, whether from sickness, vice, casualty, or old age, he is a fit and necessary subject of guardianship and protection.[1] As to what constitutes such a degree of insanity as to justify the intervention of the law in protecting the property and person of the subject and in restraining his liberty, is to be determined by the statutes and the courts in their wise discretion.[2] The rule is well settled in New York and in sister jurisdictions that if the alleged lunatic has not the capacity to conduct himself with personal safety to himself and others nor to manage his own affairs and discharge his relative duties, he is *non compos mentis*.[3]

The protection of the law being necessary to the welfare and estate of a lunatic, the question arises as to how it shall be invoked. The sacredness of property rights forbids unauthorized interference. The State being bound to protect the persons and property of those unable to care for themselves and to prevent injury to the community from the acts of those not under the guidance of reason, is the natural guardian. This is recognized universally. In England the king, being the father of his people, is vested with the care and control of incompetents, as well as orphans, minors, married women, etc. Originally exercised by the king in person, this duty was by an ordinance or standing writ of Edward III. (A.D. 1399) relegated to the Chancellor. While it is said that the powers of the Lord Chancellor in this respect are not inherent in American courts of equity, the question is one of academic interest only, as in this country the care and custody of the insane and the care of their property almost invariably devolve upon the Supreme Courts. The practical uniformity of the laws of the sister states in regard to lunatics speaks their origin plainly.

When the Constitution of 1846 merged law and equity in New York, the Chancellor's powers over lunatics passed to the Supreme and County Courts. The object of these powers is to preserve the property of the lunatic and to provide for his maintenance and support and that of his family out of his

[1] In re Barker, 2 Johns Ch., 232.

[2] Brown, Care and Custody of Incompetent Persons. [3] Idem, and cases cited.

estate. This the court cannot do personally, and it must act through an agent, called usually a committee, and in some States a guardian or curator. A committee is an officer of the court.[1] Through him the court must preserve the property intrusted to it, and therewith maintain the lunatic and his family.[2] With the various steps in the proceedings for the judicial determination of insanity and the appointment of a committee we have here nothing to do. Nearly every State prescribes the necessary procedure, and it is well to remember that any variance from the statute is inexcusable and fatal.

What a committee may do and may not do is determined by the courts and legislatures. Few branches of the law have been so satisfactorily defined by the decisions. The fundamental proposition is that a committee, either of the person or of the property, is subject to the direction and control of the court by which it is appointed. The committee is the creature of the court, enabling it to do what would otherwise be impossible.

No disposition of the real estate of a lunatic can be made by his committee, without permission first being had and obtained from a court of competent jurisdiction, and upon giving additional bonds conditioned for the faithful performance of the trust.[3] So the New York Code of Civil Procedure provides: "But a committee of the property cannot alien, mortgage, or otherwise dispose of real property, except to lease it for a term not exceeding five years, without the special direction of the court, obtained upon proceedings taken for that purpose."[4] A committee appointed by a foreign court cannot be authorized to sell property of the lunatic in another jurisdiction.[5] The fee does not pass to the committee by the fact of his appointment, for it has been held that the committee has no title or interest in the lunatic's real estate, and has no independent power to dispose of the real estate in any manner whatever.[6] Mortgages may, however, be in part released by the use of surplus funds in the committee's hands, without application to the court,[7] and surplus revenue may be used for repairs, to prevent decay and to improve the realty. On proper application the courts will restrain waste,[8] whether by the committee or another. Comparatively recent legislation in New York has given the committee power to alienate an inchoate right of dower, thus changing the former rule.[9] When it becomes necessary for any reason, such as lack of funds for the incompetent's support or that of his family, the realty may be sold. Proceedings for this purpose are regulated by statute, and must be strictly followed to avoid defect in the purchaser's title.

It is the duty of the committee to invest the incompetent's personalty. If he neglect to do so, he may be charged with interest at legal rates,[10] and if his neglect is gross, his commissions may be forfeited.[11] Should securities be accepted in lieu of cash, the committee is deemed to have made the investment himself, and will be charged accordingly. But when the trust has been administered with fidelity and with the care and caution that would characterize a prudent business man in the management of his own affairs, if loss happens, it should not be visited upon the committee.[12] Realty may be con-

[1] Code of Civil Procedure, § 2321; Matter of Dunn, 1890, 64 Hun, 18.
[2] Kent vs. West; 33 App. Div. 112.
[3] Ordronaux, Judicial Aspects of Insanity, 271.
[4] § 2339.
[5] Matter of Perkins, 1816, 2 Johns. Ch., 124.
[6] Pharis vs. Sere, 1886, 110 N. Y., 336 at p. 347.
[7] Pickersgill vs. Reed, 1875, 5 Hun, 170
[8] Matter of Hallock, 1823, 7 Johns Ch., 24.
[9] Code, § 2348.
[10] Butler vs. Jarvis, 1889, 5 Hun, 248.
[11] Matter of Gallagher, 17 N. Y., supp., 440.
[12] Matter of Hathaway, 1894, 80 Hun, 186.

verted into personalty, and the reverse, without regard to contingent interests of heirs or next of kin, provided the comfort of the lunatic be subserved, and personal property may, in the discretion of the court, be applied to the improvement of unproductive real estate.[1] Taxes and assessments must be paid by the committee, and direction from the court is not necessary to allow him to do so. Certiorari may be maintained to review the proceedings of tax boards and assessors, as the committee is the party aggrieved.[2]

Contracts made by the incompetent are not infrequently the subject of litigation. As a formal adjudication of incompetency by its very essence declares the lunatic incapable of contractural powers, it follows that any deed, contract, or agreement made after office found is absolutely void,[3] and it cannot be ratified by the committee. Where it appears that the contract was made before office found, the question is merely whether it was fairly made and without advantage being taken of the lunatic. If so, it will be sustained.[4] It has already been seen that the committee may maintain certiorari to review erroneous assessments. The committee may also maintain and defend any action, in its own name as committee, which the lunatic might have done before adjudication of incompetency.[5] The committee may also sue on a note or other negotiable instrument, and may sue to compel the payment of a legacy or distributive share. An additional bond need not be given before suit is brought, as the penalty of the committee's bond is originally fixed in contemplation of such right of action accruing.[6] Leave of the court to bring suit is unnecessary where the committee is the plaintiff; but it is contempt to sue the committee without leave first had and obtained.[7]

The helpless condition of lunatics and the greed of their relatives have resulted in many decisions fixing and determining the duty of the committee toward the heirs and next of kin. Decisions have been uniform, following the rulings of the Court of Chancery in England. Ordronaux thus states the rule:

"The first care of the court is the maintenance of the lunatic, and after that it is a rule never departed from not to vary or change the property of the lunatic so as to effect an alteration in the succession."

In the Parse Merchants' Case[8] the court said:

"No probable expense should deter the court from directing to be done whatever appeared to be most advantageous to the lunatic, *without regard to the next of kin.*"

The committee owes no duty to the heirs or next of kin except as above stated.[9] The governing principle in the management of the estate is the lunatic's interest, not that of those who have the right of succession.[10] A special term of the Supreme Court in New York county recently made an order directing the committee of a lunatic of very large estate to pay over a certain portion of the surplus income to the next of kin at stated intervals.[11] This decision does not conflict with the rule, the court presuming the incompetent would act in the same way were he sane. The application in this case was made by the committee, without opposition.

[1] Ordronaux, Judicial Aspects of Insanity; Matter of Livingston, 1842, 9 Paige, 440; Matter of Salisbury, 3 Johns. Ch., 347

[2] 1 Canady *vs.* Williams, 1895, 90 Hun, 501. Code, § 2340.

[3] Fitzhugh *vs.* Wilcox, 12 Barbour, 235.

[4] Matter of Beckwith, 1876, 3 Hun, 443.

[5] Code of Civil Procedure, § 2340.

[6] Wright *vs.* Hayden, 31 Misc., 116.

[7] Matter of Hopper, 5 Paige, 489.

[8] 3 Daly, 529; Matter of Colah, 11 Abb. Pr. N. S., 209.

[9] Matter of Reed, 18 Misc., 285

[10] Matter of Salisbury, 1814, 3 Johns. Ch., 347.

[11] Case of Ida A. Flagler.

Debts of the incompetent are to be paid and his maintenance provided for in the first instance from the personal estate,[1] but the entire estate may be expended.[2] For these purposes the income must be applied before resorting to the corpus of the estate. The committee should, by direction of the court, advertise for claims against his incompetent's estate.

Upon assuming the duties of his office the committee shall file an inventory, and he should do so whenever additional property is discovered. Some States provide for a yearly accounting and the appointment of referees to examine the condition of the trust. Final accountings occur upon the death, resignation, or removal of the committee, or the death of the ward, and involve the examination of the accounts by a referee and judicial settlement by the court. A deceased committee's administrator accounts for his intestate. Upon the death of the ward the committee's powers cease and the courts have jurisdiction only to pass on his (the committee's) accounts.

Counsel may be employed whenever legal services are necessary or whenever it is desirable to apply to the court for directions as to the investment or disposal of the ward's property. The reasonable charges thus incurred may be allowed as a necessary and proper disbursement.[3] This is so especially if the estate is large,[4] and where the estate has been benefited by an attorney's services the court will order the committee to pay for them.[5] Clerical hire may also be allowed.[6]

The committee is bound to maintain the lunatic as far as his means will allow, and to place him in such a position that he cannot injure himself or others. It is particularly the duty of the committee of the person to do this, acting, of course, under the control of the court. As before stated, the whole estate may be expended for the lunatic's support, and such costs are not to be limited by the amount of the income.[7] The extent to which the committee may go in providing for the comfort, care, and domestic establishment of the ward was carefully considered in the Matter of Reed, and the court said:

"It is the paramount duty of the committee of a lunatic to attend to his personal wants and comforts, and to furnish her, so far as the funds in his hands will allow, not only with the necessaries of life, but all the proper recreation and amusements consistent with her former habits of living. . . . The care, health, and comfort of the lunatic alone are to be considered. The maintenance of a lunatic is by no means limited to the amount of her income, but her whole estate may be expended in her support, should that become necessary. A committee may arrange for the maintenance of the domestic establishment of a lunatic to the same extent as before the beginning of lunacy."

There is no question that the committee may be authorized to provide for the keeping up of the lunatic's family establishment with the same number of domestics as had been customary previous to the lunacy, and to expend for that purpose annually an amount not exceeding that which had been annually expended before his faculties became impaired.[8] The court will act as the lunatic would were he of sound mind.[9] What constitutes a suit-

[1] Adams *vs.* Smith, 20 Abb. N. C., 50.
[2] Matter of M'Farlan, 1817, 2 Johns. Ch., 440.
[3] Matter of Clapp, 20, How. Pr. R., 385.
[4] Matter of Killick, 28 State Rep., 763.
[5] Matter of Horton, 1896, 18 Misc., 406.
[6] Matter of Livingston, 5 Paige, 440. Matter of Killick, *ante*.
[7] Ordronaux. [8] Matter of Heeney, 2 Barb. Ch., 326.
[9] Matter of Willoughby, 1844, 11 Paige, 257.

able place of confinement is a question not to be exclusively decided by the legal characteristics of the commitment. Undoubtedly the court may control the conduct of the committee in this respect, but until its power is invoked or exercised, the act of the committee will be deemed the act of the court.[1]

Compensation of the committee of the estate is properly fixed on the annual accountings, the amount being generally the same paid to executors, administrators, and guardians. In cases of more than ordinary unpleasantness or difficulty, an extra allowance will be made,[2] and so where the compensation would be inadequate.[3] The committee of the person receives an amount fixed by the court upon proper application, and is paid by the committee of the estate, and he may be allowed for personal services. When the committee of the person and of the estate is united in one person, as is customary, compensation is due only in the first capacity. An allowance for expenses is always proper.

Death of the ward or exhaustion of the estate terminates the trust, and the committee may be removed for cause or allowed to resign. Vacancies caused by resignation or removal will be filled by the court, but a committee will not be allowed to resign merely because the duties are unpleasant.

The judiciary of this country and England have invariably treated incompetent persons and their estates with the most scrupulous care. Any equities are in favor of the lunatic. No deceit or deception may be practised on him, and strict rules of law are disregarded when an injustice might result in their application. The lunatic is never properly before the court. He is not capable of consulting with counsel nor describing his affairs with accuracy or certainty. But the safeguards exercised by judges are usually sufficient compensation.

[1] Hill *vs.* Horton, 1888, 4 Demarest, 88; see also Lamar *vs.* Micou, 112 U. S., 471.
[2] Matter of Colah, ante. [3] Matter of Bayer, 1899, 57 N. Y., supp., 957.

PART II.

GENERAL PRINCIPLES OF TOXICOLOGY.

Definition.—Toxicology is the science that treats of poisons, their origin, properties, and action on the system, the treatment of their noxious effects, and their detection by chemical or other means.

Up to comparatively recent years the science of toxicology embraced in its domain only poisons of well-defined character, coming chiefly from the mineral and vegetable kingdoms, such as arsenic, strychnin, and morphin. Within a relatively short time, however, it has been discovered that a large number of diseases are produced by the action of certain poisonous substances generated within the system either by the normal cells of the body or by micro-organisms. These compounds, known according to their origin and properties as leukomains, ptomains, toxins, etc., properly come within the scope of toxicology and give to the science a greatly increased field. It naturally is not customary, however, to deal with these substances in works like the present on forensic toxicology, but rather to leave their discussion to books on pathology and the practice of medicine, or to treatises devoted especially to them.

It is a difficult matter to give an exact definition of the term "poison." This arises from the fact that it is impossible to set aside any group of substances that are poisons under all conditions; a body may be entirely harmless under certain circumstances, and yet under others be possessed of dangerously poisonous properties. The salts of potassium, for example, in small quantity are not only not poisonous, but are necessary for the maintenance of a healthy condition of the body; in large doses, however, they are active poisons, capable with great certainty of producing death. Most medicines if administered in too large quantity become poisons; and conversely, nearly, if not quite all, poisons, if given in small doses, are possessed of remedial properties. The line, in fact, between a medicine and a poison is often exceedingly narrow. No substance is known that is a poison in all doses; a certain amount, varying, however, very widely with different bodies, is necessary in order that poisonous effects may follow its administration.

The definition given by Taylor[1] is, when slightly modified, perhaps as comprehensive and as scientific as any:

A poison is a substance which, when absorbed into the blood, is capable of seriously affecting health or of destroying life, and this as its usual effect upon the healthy body.

It will be observed from the above—

[1] *On Poisons*, p. 18.

First, that a poison is a material substance, and not an imponderable agency, such as electricity or heat.

Second, that it is a body which acts after absorption into the blood, and does not produce its effects, therefore, by mere mechanical action. Pounded glass, needles, and other similar articles, although capable of producing death when taken in sufficient quantity, act mechanically only, and are not poisons in the true sense of the word. For similar reasons, it may be a question whether the corrosive acids and alkalis should be considered as poisons. Their chief effect is a local one and is produced without absorption into the blood, and on this account some writers[1] on toxicology do not embrace them under the strict head of poisons. Since, however, in nearly all cases a certain part of the corrosive agent enters the circulation and produces harmful systemic effects, their exclusion from the category of poisons seems on the whole unnecessary, and for many practical reasons undesirable.

Third, that a substance which produces noxious effects as an unusual result, or by acting upon a diseased body, is not necessarily a poison. Many ordinary articles of food, as is well known, are occasionally the cause of distressing and sometimes even of serious symptoms when taken by people who have an idiosyncrasy to them. Strawberries cannot be eaten by a considerable number of people without unpleasant effects,[2] and the writer is acquainted with a gentleman in robust health who is made exceedingly ill not only by eating apples, but even by their odor. Strawberries and apples, however, manifestly should not come within the definition of a poison; nor do they, as the term is defined above, as the noxious results noted are not their *usual* effects. And similarly, improper articles of food, which have more than once produced fatal effects with typhoid patients, are equally excluded by our definition from the category of poisons.

Fourth, that whether a substance is a poison or not is in no way dependent on the quantity that must be used to produce noxious results. Half a grain of strychnin may produce death, while sixty grains of oxalic acid are required to occasion fatal results.

Although from a strictly scientific standpoint the size of dose necessary to produce noxious effects has nothing to do with the conception of a poison, yet, as commonly understood and as generally accepted in ordinary life, the term "poison" is closely connected with the amount required to induce serious results. Common salt in massive doses is capable of producing noxious effects, and has even occasioned death,[3] but it is not commonly looked upon or spoken of as a poison, the quantity required for these results being so large. We may say, perhaps, in a general way that in the everyday affairs of life a substance to be regarded as poisonous must be capable of inducing

[1] Witthaus and Becker, *Medical Jurisprudence, Forensic Medicine, and Toxicology*, vol. iv., p. 43.

[2] Gould and Pyle, *Anomalies and Curiosities of Medicine*, p. 489, where several cases of illness and one of death from the eating of strawberries are cited.

[3] *Medical Times*, January 4, 1840, p. 133 (referred to in Woodman and Tidy's *Forensic Medicine and Toxicology*).

harmful results if administered in doses of about sixty grains, if a solid, or a teaspoonful if a liquid. This limitation, although somewhat arbitrary, is often of great convenience in the common use of the word "poison" and in the discussion of the subject in medicolegal cases.

In order to avoid any uncertainty as to the inclusion of the corrosives and the mechanical irritants, the statutes commonly speak of "the administration of a poison or of other noxious or destructive thing," or use other words to the same effect. Legal quibbling in regard to definitions is thus eliminated or reduced to a minimum.

Classification of Poisons.—A large number of classifications of poisons have been suggested, but none of them is entirely satisfactory. Two general systems of classification have usually been adopted, the first depending upon the origin or nature of the substance; the second, upon the effect it produces on the system.

Were our knowledge of the actions of poisons absolutely definite, the second system of classification would unquestionably be the most desirable; but since, in the present state of science, our information on this point is by no means complete, any close classification made upon this line is necessarily faulty. A simple, general division of poisons, however, into **corrosives, irritants,** and **neurotics,** according as the chief effect produced is local corrosion, gastro-intestinal irritation, or altered action of the nervous system, is often exceedingly useful, and is the one I prefer if the classification is based on the effects shown. In this classification under *corrosives* are included the strong acids and alkalis, whose most important action is local destruction of the tissues; under *irritants*, those numerous substances, such as arsenic, antimony, mercury, and most of the other heavy metals, phosphorus, bromin, iodin, and a certain number of organic substances like cantharides, savin, and croton oil, whose most conspicuous effects are usually gastro-intestinal irritation, as shown chiefly by vomiting, purging, and local pain; and under *neurotics*, the majority of organic poisons, prominent among which are alcohol, chloral, chloroform, opium, belladonna, aconite, strychnin, acetanilid, carbon monoxid, hydrocyanic acid, and carbolic acid, all of which expend their toxic powers chiefly on the nervous system, producing delirium, coma, convulsions, and disordered circulation and respiration as prominent symptoms.

Even this division, however, is seriously deficient and far from satisfactory, as very many poisons act in two or more different ways: oxalic acid in concentrated solution is a corrosive, in dilute solution an irritant, and in either case it also acts powerfully on the nervous system, and the same may be said of mercuric chlorid, carbolic acid, and numerous other poisons.

As an interesting contribution to this system of classification we give below the grouping of poisons suggested by Rabuteau.[1] It is faulty, as any classification on this line must necessarily be in the present state of our knowledge, but is suggestive of what may finally

[1] *Eléments de toxicologie*, p. 31.

be done in this direction when further research has more fully informed us of the exact mode of action of our various poisons.

I. HEMATICS.

1st. Those acting specially on the red corpuscles: Carbon monoxid, hydrocyanic acid, hydrogen sulphid and ammonium sulphid, phosphorus, the alcohols, and the compounds of arsenic, selenium, and tellurium.

2d. Those acting on the corpuscles and the plasma: Nitrites and nitrous vapors, salts of silver injected into the veins, the majority of metallic salts (in small and continued doses).

II. NEUROTICS.

1st. Motor paralyzers, abolishing the functions of the motor nerves: Curara, Calabar bean, aconitin, and coniin.

2d. Spinants, exaggerating reflex action: Strychnin, akazga, compressed oxygen, cantharides, etc.

3d. Cerebrospinants, acting on the brain and spinal cord: Chloroform, ether, and opium.

III. NEUROMUSCULAR.

Solanaceæ, digitalis, and compounds of antimony.

IV. MUSCULAR.

Carbon dioxid, strophanthus, veratrin, and salts of potassium, barium, copper, zinc, cadmium, tin, lead, mercury, etc.

V. IRRITANTS AND CORROSIVES.

Sulphuric, nitric, hydrochloric, hydrofluoric, oxalic acids, potassium and sodium hydroxids, ammonia, alkaline sulphids, iodin, bromin, chlorin, etc.

After mature consideration of the subject I finally agree fully with Chapuis,[1] that under present conditions the most useful classification of poisons for forensic purposes is that depending on their origin or nature, and consequently, in the present book, the division adopted is the following: First, Inorganic Poisons; Second, Alkaloidal Poisons; Third, Non-alkaloidal Organic Poisons; Fourth, Gaseous Poisons; and Fifth, Food-poisons. This classification commits us to no theory regarding the precise action of a poison, and at the same time it brings together those of a similar nature from an analytic standpoint and possessed otherwise of many points in common—items of much moment in forensic toxicology to all concerned.

CONDITIONS AFFECTING THE ACTION OF POISONS.

The effects of poisons on the system are often very materially modified by a number of circumstances, and these may be conveniently divided into two groups: I. Those relating to the poison itself and its administration; and II. Those relating to the individual.

I. Under the first heading we have two chief subdivisions: 1. The form of the poison; and (2) the avenue of its entrance into the circulation.

1. **Form of the Poison.**—We have seen that, in order that a

[1] *Précis de toxicologie*, third edition, 1897, p. 119.

substance may act as a poison, it must be capable of entering the blood; in other words, it must be a soluble body. It is evident, therefore, that no substance can be a poison that is entirely insoluble. Other things being equal, a given poison is more active the more soluble the form in which it is administered; barium, for instance, when combined with chlorin, as the readily soluble chlorid, is highly poisonous, but when united with sulphuric acid, as the insoluble sulphate, it is destitute of toxicity. This general principle is extensively taken advantage of in antidoting poisons, as will be seen later.

Poisons in the form of a solid generally produce their effects more slowly and less energetically than when taken in solution; in pills and capsules their action is likely to be particularly retarded. In these latter forms they may pass through the intestinal tract incompletely or irregularly dissolved, and possibly occasion, therefore, unexpected results. The case cited on page 307, in which the symptoms of arsenical poisoning did not appear until the sixteenth hour, undoubtedly owed the long delay chiefly to the fact that the poison was taken in the form of a dry powder and without fluid to wash it down. Pashley [1] relates a remarkable case in which a healthy young man swallowed in pills three grains of strychnin, a dram of opium, and a considerable quantity of quinin, and no characteristic effects were developed until twelve hours had elapsed; coma then supervened and the man died a number of hours later, without, however, at any time showing symptoms indicating the action of strychnin.

In this connection it may be stated that poisons when taken on a stomach full of solid food are generally more slowly dissolved and less rapidly absorbed, and, as a consequence, produce their effects somewhat more tardily and frequently less severely than if taken when the stomach is empty or contains but little food. The importance of this fact in forensic toxicology is often great, as it explains at times many seeming anomalies in the action of poisons.

Dilution of a poison by water to a certain extent, by favoring its more rapid absorption, usually hastens and intensifies its action. Corrosive poisons, however, as would be expected from their very nature, have their activity greatly diminished by dilution.

2. **Avenue of Entrance into the Circulation.**—As a rule, poisons produce practically the same effect by whatever path they enter the blood, the only difference being due to the different rates of absorption. Administration by inhalation, by hypodermic injection, or by direct injection into the blood-current causes generally much more rapid and energetic symptoms than when the poison is given by the stomach or rectum; but otherwise the effects are usually identical or not greatly different. In a few cases, however, the avenue of entrance materially modifies the action. Thus, snake-poisons when given by the mouth are entirely harmless; and curara is toxic in only a very slight degree when administered by the stomach, although intensely poisonous if given hypodermically. Hydrogen sulphid, even in small amount, is extremely

[1] *Chicago Med. Jour.*, November, 1860, p. 625.

poisonous when inhaled into the lungs, but the same quantity may be administered by the rectum, or, when in solution, by the mouth, without danger. Chloroform, ether, and a number of other analogous substances occasion effects quite different in some respects when administered by the mouth from those produced when given by inhalation.

II. The chief conditions relating to the individual that modify the effect of poisons are: 1. Age; 2. Idiosyncrasy; 3. Habit; 4. Tolerance; 5. Disease.

1. **Age.**—As would naturally be inferred, the age of a person decidedly affects the susceptibility to poisons. The general rule is that the younger the person, the greater the susceptibility. There are, however, some exceptions to this, young children, for example, being far more tolerant of calomel and belladonna than adults. But, on the other hand, certain poisons act upon infants with unexpected severity; and this is especially true of opium and its preparations, and of narcotic drugs in general, all of which are greatly more poisonous proportionally to young children than to the adult. One or two drops of laudanum have proved fatal to infants, and Taylor [1] relates a case in which an infant four weeks of age died presumably from the effects of a quantity of paregoric equivalent to only one-ninetieth of a grain of opium.

2. **Idiosyncrasy.**—Many persons manifest an unusual, and in most cases inexplicable, sensitiveness to the action of certain poisons, and this fact has not infrequently been the cause of serious disturbance, and even of death, from the remedial administration of these substances in the practice of medicine. Marked individual intolerance of antipyrin, of cocain, of even such comparatively harmless substances as quinin, and, in fact, of nearly every known drug and of many articles of food, is almost daily observed by the busy medical practitioner. In all cases of poisoning the possibility of the existence of this unusual sensitiveness should be borne in mind.

Idiosyncrasy is presumably the cause also of certain rare cases in which poisons produce very unusual symptoms or even entirely opposite effects from those generally observed. Shearman [2] reports a case in which a grain and a half of morphin was followed not by coma, but by convulsions resembling those caused by strychnin; and Philips [3] records an instance in which atropin caused contraction of the pupils instead of dilatation. Such cases are naturally likely to occasion mistakes in diagnosis and lead to much confusion in the event of a judicial inquiry; and their possibility, therefore, should always be borne in mind in forensic investigations.

3. **Habit.**—By the continued use of almost any poison, beginning with small doses and gradually increasing them, most persons may become habituated to their effects, and finally are able to take large quantities without the manifestation of toxic symptoms. This is shown in the case of habitual morphin-takers, many of whom, as is well known, are able to take doses that would prove rapidly fatal to those unaccustomed

[1] *On Poisons*, p. 536. [2] *Medical Times and Gazette*, March 7, 1857, p. 235.
[3] *Ophthalmic Record*, January, 1903, p. 5.

to its action. De Quincey, according to his *Confessions*, consumed each day a quantity of laudanum equivalent to about 360 grains of opium, and even this quantity of the narcotic has been greatly exceeded in more recently recorded cases. Russell[1] reports a case in which 60 grains of morphin were used daily hypodermically for some time, after which, for several months, 30 grains each of morphin and cocain were taken per diem. The arsenic-eaters of Styria, in Austria, it has been abundantly demonstrated, take habitually such quantities of arsenic as would be fatal to the unhabituated, long use of the poison from early years having accustomed them to its effects. Van Fleet[2] describes a case of alcoholic amaurosis in which strychnin was administered hypodermically in gradually increasing doses until the patient received two-thirds of a grain twice a day, and this was kept up for a long time with marked benefit to the general health.

The tolerance of poisons gained by habit is, however, only relative; a sufficient increase of dose beyond the accustomed limit develops the usual toxic powers of the substance, and many cases are recorded of death among morphin habitués from quantities of the drug exceeding the amount to which they were habituated. At least one instance of this has come under my own observation. A woman of middle age, who for several years had been accustomed to take by the mouth from two to four grains of morphin two or three times a day, swallowed, either by mistake or with suicidal intent, not less than ten or fifteen grains of the drug. She soon fell into a profound stupor, exhibited all the usual symptoms of morphin-poisoning, and died at the end of eighteen hours.

4. **Tolerance.**—Occasionally persons are found who manifest a remarkable natural tolerance of the action of certain poisons that is wholly independent of habituation. The author is acquainted with a physician who, although entirely unaccustomed to its use, is able to take a grain of sulphate of morphin without the production of the slightest apparent effect. Christison[3] relates a similar but even more remarkable case of a man, unaccustomed to the use of opium, who took nearly an ounce of laudanum without any effect. Many other illustrations of similar tolerance are on record. No explanation as yet has been afforded of this phenomenon.

5. **Disease.**—The action of poisons is not infrequently considerably modified by disease, their effects in some cases being materially increased, and in others greatly diminished. Organic diseases of the kidney, by retarding elimination, as a rule decidedly increase the effects of most poisons, a dose that would ordinarily not be overlarge sometimes producing serious results when the person is suffering from renal disorder. It should be stated, however, that the recent experiments of Meltzer and Salant[4] and of Meltzer and Langmann[5] seem to throw

[1] *Medical Record*, N[illegible]
[2] *M*[illegible] *E*[illegible]
[3] *Treatise on P*[illegible] *M*[illegible] Feb., 1902, p. 167.
[5] *Journ. of Med. Research*, F[illegible]

some doubt on this commonly accepted doctrine. Operating on nephrectomized rabbits and guinea-pigs, they found that strychnin was fully as well borne by them as by normal animals.

On the other hand, delirium tremens, tetanus, and diseases attended with great pain, such as peritonitis and the passage of gall-stones, give the system a remarkable tolerance to anodynes like morphin, chloral, and chloroform. In these conditions doses of the anodyne may generally be administered which in a healthy adult would produce serious, or even fatal, results.

DIAGNOSIS OF POISONING.

The diagnosis of poisoning before death is of the greatest importance in order that proper treatment may be directed, and also that accurate records may be made of the condition of the patient to be used in case of subsequent legal inquiry. Unfortunately, however, the diagnosis of poisoning is usually exceedingly difficult, and frequently impossible, in the living subject. This arises from the fact that, with one exception, the symptoms produced by poisons are not clearly characteristic, and cannot by themselves alone be distinguished with absolute certainty even by the most experienced observer from the symptoms of disease. The one exception referred to is that of strychnin, whose effects on the living subject, when typically shown, are so entirely characteristic as generally to leave no doubt as to their cause.

The **diseases simulating poisoning** are many, those resembling *irritant poisoning* being chiefly cholera, cholera morbus, acute indigestion, ulceration of the stomach, gastritis, gastro-enteritis, peritonitis, appendicitis, hepatic and possibly renal colic, and intestinal obstruction ; while those whose symptoms resemble *poisoning by neurotics* are, among others, hysteria, cerebral hemorrhage and thrombosis, epilepsy, the convulsions of certain organic brain diseases, tetanus, inflammation of the brain and its coverings, uremia, and organic heart disease.

The careful consideration in connection with the symptoms, of certain concomitant circumstances or conditions will often enable a physician to arrive at a conclusion of presumable accuracy, and sometimes to make an indisputably correct diagnosis. The presence on the lips and tongue and in the throat of marks of corrosion almost of necessity lead to the conclusion that a corrosive poison of some kind has been used, and the odor of the breath frequently discloses unerringly the administration of carbolic acid, chloroform, and the preparations of crude opium. The careful examination of vomited matter, and sometimes even of the feces, occasionally gives important evidence ; the odor of carbolic acid, of potassium cyanid, and of laudanum, the luminosity in the dark of phosphorus, and the sparing solubility and crystalline character of particles of arsenic trioxid afford valuable aid in making a diagnosis. In the absence of these indications, and sometimes even in their presence, the following rules are frequently of great value in distinguishing poisoning from disease :

I. In cases of poisoning the symptoms usually appear suddenly and generally when the patient is in health.

II. In cases of poisoning the symptoms commonly make their appearance after the taking of food, drink, or medicine.

III. If several persons take the same food or drink, they all show similar symptoms.

Each of these points deserves separate consideration.

I. Diseases, as a rule, do not appear suddenly in persons in health, but usually are preceded by a number of hours, days, or even weeks of general or local indisposition; poisons, on the other hand, generally produce their symptoms suddenly, striking down persons in health with great severity and with but a short period of antecedent distress.

It should be remembered, however, that there are important exceptions to this rule on both sides. While nearly all poisons begin to show their effects, in the great majority of cases, within an hour or two at most after their administration, and, some, like the corrosives, hydrocyanic acid, and strychnin, usually either almost immediately or within a few minutes, still many cases are recorded in which the full symptoms of the poison have been delayed a number of hours. Hartshorne[1] relates a remarkable instance of poisoning by a dram of arsenic trioxid in which the symptoms did not appear for sixteen hours; Christison[2] records a case in which upward of two ounces of tincture of opium was taken and no well-defined symptoms showed themselves till the eighteenth hour; and a case is reported[3] in which a woman ate a considerable quantity of aconite root, and no characteristic effects developed until ten hours afterward. The form in which the poison is administered and the condition of the stomach as to whether containing food or not are important factors, as we have already seen, in determining the rapidity of absorption and consequently the time of the production of symptoms.

On the other hand, diseases sometimes manifest themselves with great suddenness in persons enjoying apparently perfect health, as is seen occasionally in cases of apoplexy, heart disease, obstruction of the bowels, etc.

In cases of chronic poisoning, in which small but frequently repeated doses are administered, the physician is very easily misled, as the symptoms under these circumstances resemble those of disease more nearly than when a single large dose is given. So, too, when in the progress of a disease a poison is administered, whether in one large or in several small doses, the diagnosis is rendered doubly difficult.

II. Sudden sickness setting in soon after taking food, drink, or medicine, especially if the symptoms are aggravated by taking additional quantities of the substance, is very suggestive of poisoning, and should always put a physician or attendant on his guard. Under these circumstances a careful examination of the suspected article will occasionally give important confirmatory proof by the taste, odor, altered general appearance, or presence of foreign particles. But it should be borne

[1] *Medical Examiner*, December, 1855, p. 707. [2] *Treatise on Poisons*, p. 544.
[3] *The Lancet*, October 6, 1860, p. 344.

in mind that some diseases, such as acute indigestion, apoplexy, and perforation of the stomach, are prone to manifest themselves after eating or drinking, and that a number of other maladies are not infrequently increased in severity by taking food or drink.

III. If a number of persons who have taken the same food or drink manifest similar symptoms, the evidence of poisoning is usually quite strong. Suspicion of poisoning, however, should not of necessity be dismissed from the mind even if but one person shows symptoms of it and others who have partaken of the same food or drink escape, for it is quite possible that the poison may have been introduced into only a single portion of the food, or by chance that the other person may have a tolerance more or less complete of the poison used. Numerous instances of these kinds, especially of the first, have been recorded. In the case of the State of Wisconsin *vs.* Zoldoske[1] it was clearly shown that the deceased woman came to her death from strychnin taken in a piece of candy, although a number of other persons who ate candy from the same box experienced no ill effects.

While a careful observance of the above rules and a critical study of the symptoms presented frequently enable the physician to make a reasonably reliable working diagnosis, yet in order to distinguish poisoning from disease with absolute certainty during life a chemical analysis of the food, drink, or medicine taken, or, better still, of the vomited matter and urine, is generally necessary. Unfortunately, in the majority of cases this is impracticable, the length of time necessary to make an accurate analysis for most poisons, and the expense attending it, being too great. But for arsenic, mercury, and antimony we have in Reinsch's test a rapid, easy, and reliable means of detection, which every physician should be prepared to perform. For a description of this test see p. 327.

TREATMENT OF POISONING.

The treatment of poisoning may be divided into four parts: I. The removal of the poison from the stomach; II. The administration of antidotes; III. The elimination of the poison; and IV. Counteracting the constitutional effects of the poison and sustaining the system.

I. The removal of the poison from the stomach may be accomplished either by inducing vomiting, or by the use of the stomach-pump or stomach-tube. If, as is often the case, vomiting is already present, it is usually sufficient simply to encourage it by the administration of copious draughts of tepid water, with which preferably some greasy substance, like oil or lard, has been mixed. If, however, vomiting has not set in, or if it is not sufficiently copious, means should be used to produce it. Thrusting the fingers into the back of the mouth and depressing the tongue, or tickling the fauces with a feather, are commonly known procedures for inducing vomiting, but the surest means is the use of an emetic. Of these there are many, but perhaps the most useful are a dessertspoonful of ground mustard stirred up in a

[1] 82 Wisconsin, p. 580.

small cup of tepid water, fifteen or twenty grains of zinc sulphate, ten or fifteen grains of copper sulphate, or, what is generally best of all, a tenth of a grain of apomorphin hydrochlorate administered hypodermically. Whatever method is employed, the vomiting should be kept up and the stomach thoroughly cleansed by the administration, as before described, of abundant draughts of tepid greasy water, in which appropriate antidotes may also be placed. Water in which dishes have been washed (dish-water) is often particularly useful to encourage vomiting—its greasy character, tepid temperature, and nauseating taste and odor make it especially efficient whenever its slight alkalinity, due to the presence of soap, is not objectionable.

The stomach-pump (see Fig. 22) and stomach-tube are usually to be preferred to emetics, both because they produce less prostration and generally are somewhat more efficient in completely removing the poison. The stomach-tube, being much more frequently at hand, is more commonly used than the stomach-pump. Its introduction is usually easily accomplished. The patient, if able to assist, should be seated and lean the head a little forward; artificial teeth should be

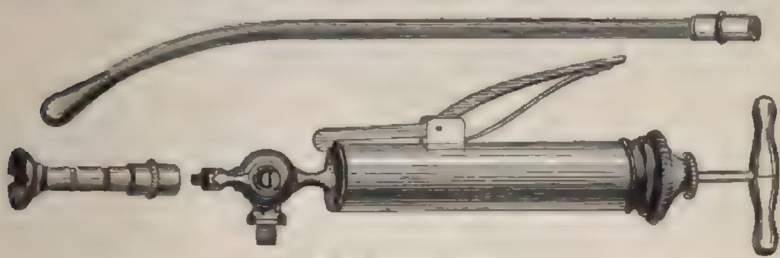


FIG. 22.—Stomach-pump.

removed and tight clothing loosened. The tube is carried to the back of the tongue and the patient asked to swallow. As he does so, the tube is gently pushed into the esophagus and, while the patient breathes regularly and makes occasional efforts at swallowing, it is passed by very moderate pressure into the stomach. In the absence of a stomach-tube a piece of ordinary rubber gas-tubing with a funnel inserted in one end can be made to take its place. As a rule, a pint of water should at once be introduced into the stomach through the tube and removed, and the operation repeated four or five times, or until it is evident that the contents of the stomach have been efficiently washed out. Antidotes may generally advantageously be placed in the water, as a portion almost surely remains in the stomach and delays the action of the poison.

The stomach-pump, if at hand, is somewhat to be preferred to the tube, as it affords a surer means of emptying the stomach, especially if the latter contains a considerable amount of undigested food; but the effect of the pump can be obtained nearly as well by the use of the tube if provided with a bulb (see Fig. 23).

However the stomach is emptied, the contents should be received

in perfectly clean bowls or buckets and carefully inspected to ascertain if any poison recognizable by its physical properties is present. The odor of carbolic acid; luminous particles of phosphorus; heavy, white, sparingly soluble grains of arsenic trioxid (arsenious acid), etc., are frequently important guides in determining the exact character of the poison used and in directing appropriate treatment. It is desirable in all cases to save a part or all of the material ejected for subsequent chemical analysis, in case of legal inquiry.

In poisoning by the corrosive acids and alkalis, it is generally considered inadmissible by most authorities to use either emetics or the stomach-tube, on account of the danger of producing perforation of the stomach or esophagus from softening of these parts by the corrosive agent. In this form of poisoning it is commonly recommended that reliance be placed on copious dilution by water or milk and the use of appropriate chemical antidotes.

Hemmeter,[1] however, differs with most of the writers in regard to this matter, and I believe with reason. He says: "In six cases of poison-

FIG. 23.—Soft-rubber stomach-tube, with funnel and exhaust bulb.

ing—one with lye, two with oil of vitriol, one with strong ammonia, and two with carbolic acid—the tube was used immediately after the patients reached the hospital. As such cases run great danger of a corrosive perforation we have personally used the tube and let the patient take his chances, which were better in these cases than in those where the tube was not used." He says further: "We make so explicit a statement of treatment because we had experience with two cases where the autopsy showed that recovery might have been possible (as not much sulphuric acid had reached the stomach) if the treatment had been more heroic—*i. e.*, if the tube had been used for the timely removal of the poison."

II. **Antidotes** are of two kinds: 1. Those that act mechanically; 2. Those that act chemically.

1. Antidotes that act *mechanically* are either those that envelop the poison in some insoluble material, or those that coat the mucous membrane of the stomach in a similar manner; in either case the result being a very greatly reduced rate of absorption of the poisonous body. The

[1] *Diseases of the Stomach*, second edition, 1900, p. 443.

most important of these substances are fats and oils, milk (on account of the coagulum of casein formed in the stomach), the white of eggs, and charcoal in a moderately fine state of subdivision. The latter acts by absorbing the poison within its pores, and is especially efficient in connection with the organic poisons. Fats and oils are of very wide utility, both on account of materially retarding the absorption of the poison, and also by protecting the mucous membrane of the stomach from corrosive or irritant action; in case of poisoning by phosphorus, however, they are entirely inadmissible, as they dissolve the poison and render it much more active, and, for the same reason, milk is undesirable unless it has been well skimmed. It is sometimes stated that milk is also inadmissible in poisoning by arsenic trioxid, its alkalinity favoring solution of the poison, but this objection is not well grounded, since the reaction of cows' milk is practically always feebly acid, at least to phenolphtalein.[1]

2. The proper *chemical antidote* for each poison will be given later on in the discussion of the individual poisons, and only a few points of general interest need be considered here.

The majority of antidotes that act chemically do so by reacting with the poison to produce an insoluble or sparingly soluble compound, and one, therefore, that is either inert or of only slight toxicity. The use of common salt as an antidote in poisoning by silver nitrate is an illustration of this, insoluble and consequently harmless silver chlorid being produced by the chemical action of the antidote upon the poison. Some, however, act by changing the chemical character of the poison without the production necessarily of an insoluble body, as is the case when acids are used to neutralize caustic alkalis, and when potassium permanganate is administered as an antidote to morphin. It should always be borne in mind in the use of chemical antidotes, in whatever way they act, that the compounds produced are often only relatively harmless, and that if allowed to remain in the stomach for some time serious results may be produced; thus, ferric hydroxid when used to antidote arsenic unites with the latter, producing a compound of very sparing solubility in pure water, but as it is somewhat soluble in dilute acids, it is liable to be dissolved and the poisonous properties of the arsenic redeveloped if allowed to remain in the stomach for some time. In all cases, therefore, it is well after the administration of an antidote to empty the stomach, repeating the operation a number of times.

In the selection of a chemical antidote a substance should always be chosen, when practicable, which is by itself as nearly harmless as possible, so that if an excess is given it will do little or no damage. Thus, in choosing an acid to antidote a corrosive alkali, vinegar or lemon-juice are greatly preferable to sulphuric or hydrochloric acid, which, if given in too large quantity might occasion as much injury as the original poison.

If a case of poisoning is seen late, and especially if free vomiting has already been established, it is often assumed that no poison remains

[1] Duclaux, *Principes de laiterie*, p. 27.

in the stomach, and that consequently antidotes are unnecessary. Such, in fact, is often the case, but it is by no means always so. Antidotes, whether acting chemically or mechanically, should not only be given as early as possible, but their administration should be kept up until it is absolutely certain that no more of the poison remains in the stomach. Some poisons, especially arsenic in the form of the trioxid, are prone to persist in the stomach for many hours, frequently in spite of profuse vomiting. In a case examined by the writer, in which death occurred eighteen hours after the administration of a teaspoonful of arsenic trioxid, and in which copious vomiting had continued for several hours, a fraction more than six grains of the poison were found in the stomach after death, largely in the undissolved form, adhering closely to the mucous membrane. In another case in which death took place forty-eight hours after an unknown quantity of arsenic trioxid had been taken for suicidal purpose, nearly two grains of the undissolved poison were found in the stomach. Profuse vomiting had occurred soon after the poison was taken, and the physician who was called in twelve hours later thought it unnecessary to wash out the stomach or give antidotes, unfortunately feeling convinced that all the poison had been necessarily removed by the abundant vomiting.

It must be admitted that it is sometimes extremely difficult to determine when all the poison has been removed from the stomach, and when antidotes, therefore, may be safely discontinued; the use of Reinsch's test, however, in case of poisoning by arsenic, antimony, or mercury, gives us conclusive information, and the odor of the vomited material is of great service in arriving at correct conclusions when carbolic acid or other poisons of characteristic odor have been taken. In poisoning by opium and its derivatives it is especially important to continue the use of antidotes, as it is well known that however these poisons are administered, their poisonous morphin is eliminated to a considerable degree by the mucous membrane of the stomach; washing out that organ, therefore, and the administration of antidotes are serviceable at almost all stages.

Universal Antidote.—In case no information is at hand in regard to the nature of the poison taken, it is evident that the exact antidote cannot be administered. Under these conditions, however, it is not infrequently highly advantageous to give a combination of two or more substances of a harmless character which are efficient at least in a moderate degree in antidoting a number of poisons. Several combinations of this character have been suggested. Perhaps none serves the purpose as well as a mixture of pulverized charcoal, tannic acid, and magnesia, using about two parts of the first to one part each of the other two. The mixture may be given in doses of a heaping teaspoonful stirred up with water and may be frequently repeated, as none of the constituents is harmful. The charcoal acts physically, taking up alkaloids and many other poisons into its pores, greatly retarding their absorption by the system; the tannic acid precipitates alkaloids and many of the metals; and the magnesia neutralizes acids, and, next to

ferric hydroxid, is probably our best antidote for arsenic. The mixture, consequently, antidotes more or less efficiently a wide range of poisons and, somewhat questionably however, has received the appellation "universal antidote." It should not be used to the exclusion of more thoroughly appropriate antidotes when the exact nature of the poison is known; and with this, as with all other antidotes, vomiting should be encouraged or the stomach washed out shortly after the administration of each dose, except a final one, which is preferably left in the stomach.

Physiologic Antidotes.—The antidotes we have been considering act either mechanically or chemically; but there is another class of bodies often used in the treatment of poisoning which produce their effect purely by physiologic action, and hence have been designated "physiologic antidotes." Such are atropin in the treatment of poisoning by morphin, and chloroform in strychnin-poisoning. The term physiologic antidote, however, has been objected to, and perhaps justly, for such substances do not truly neutralize the poisons for which they are given, but simply antagonize their effect and that, too, only in part. On this account the more appropriate term for them is undoubtedly *physiologic antagonists*. Probably the best example of physiologic antagonism is that shown by atropin and physostigmin. Atropin accelerates the heart's action while physostigmin slows it; atropin dilates the pupil while physostigmin contracts it; atropin arrests glandular secretion while physostigmin increases it; atropin stimulates, while physostigmin depresses the central nervous system. In most other cases, however, the antagonism is usually limited to only one or two of the effects produced, their action in other ways not infrequently being synergistic. Thus atropin increases the reflex function of the central nervous system while morphin lessens it, and consequently the former in small doses may be used with advantage as a partial physiologic antagonist in the treatment of opium-poisoning; but in other respects there is little or no real antagonism—in fact, their toxic action in certain ways coincides, and consequently if atropin is administered in opium-poisoning it should be only in limited quantity.

Among other physiologic antagonisms may be mentioned atropin to pilocarpin, strychnin to nicotin, digitalis to aconite, chloral to strychnin, caffein to morphin, and each of them has been used in the treatment of poisoning by the other.

III. In the majority of cases of poisoning it is the poison that enters the circulation that produces most or all of the harmful results, and it is exceedingly important, therefore, that **elimination** should be encouraged, so that the dangerous agent may be removed from the body as soon as possible. The experiments of Claude Bernard, showing that if the elimination and the absorption of a poison are kept equal harmful results do not appear or are reduced to a minimum, are of great interest in this connection.

The chief emunctories by which poisons are eliminated are the kidneys, skin, and bowels, and all of these, therefore, should be stimulated to their fullest activity. The kidneys are of special importance,

as in the majority of cases they are the chief avenue of elimination of most poisons. Diuretics, consequently, should be exhibited, and of these perhaps the best is pure water copiously administered after the poison has been removed from the stomach; and small doses of potassium acetate, digitalis, diuretin, or other similar diuretics may be added, if not otherwise contraindicated. Hot applications over the region of the kidneys will sometimes stimulate them to activity, especially when they are congested by the irritant influence of the poison. The urine should not be allowed to accumulate in the bladder, but should be drawn off by a catheter from time to time, if the patient is unable to void it naturally. The bowels, if inactive, should be emptied by enemas of tepid water or by the administration of mild laxatives, and the skin should be kept active preferably by warmth, but medicinal diaphoretics, such as pilocarpin given hypodermically, are sometimes of great utility, especially if the kidneys persistently refuse to perform their office.

The success obtained in certain diseases of toxic origin, such as puerperal eclampsia and other forms of uremia, by the hypodermic injection of normal salt solution (hypodermoclysis), has led to the application of the same measure to the treatment of poisoning. The remedy acts by diluting the poison in the blood and by favoring the action of the eliminative organs, especially of the kidneys. The operation is performed by attaching an ordinary irrigator to a large hypodermic needle, taking care that the liquid used and everything about the apparatus are thoroughly sterile. The solution can be made with sufficient accuracy by dissolving a heaping teaspoonful of common salt in a quart of boiling water. Sodium carbonate and certain other salts have at times been used to replace, in part, the sodium chlorid. It should be administered at a temperature of about 110° F., and a quart is generally used, although half or a quarter of this quantity is sometimes to be preferred. In cases where some delay in its action is permissible the warm salt solution may advantageously be administered by the lower bowel (enteroclysis), as there is then no danger of infection from imperfect sterilization. On the other hand, in case of poisons that act with great rapidity we may even resort to the intravenous injection of the saline solution.

Several cases have been recorded showing the great value of the above treatment by saline infusion. Typical among them I may refer to the case reported by Hirsch and Edel[1] of phenyl-hydroxylamin poisoning with recovery, after the use of stimulation and venesection followed by the subcutaneous injection of a liter of a solution containing 0.3 per cent. of sodium chlorid and 0.4 per cent. of sodium carbonate; and to a case of poisoning by 8 grains of morphin taken hypodermically reported by Willoughby,[2] in which after strychnin, atropin, and other measures had been unsuccessfully tried, the infusion of a liter of salt solution in the flank led to a rapid recovery.

IV. In most cases it is the **systemic effects of poisons** that

[1] *Berlin. klin. Wochenschr.*, Oct. 14, 1895, p. 891.
[2] *The Lancet*, May 10, 1902, p. 1316.

are chiefly dangerous, and these, therefore, as well as the local action, should be combated from the beginning. Anodynes should be administered for the control of excessive pain; strychnin, nitroglycerin, and digitalis given for threatened heart failure; and artificial respiration or faradization of the phrenic nerve used in the event of failure of respiration. Inhalations of pure oxygen are often of service in case of deficient oxygenation of the blood, and chloral or chloroform is of great value in controlling convulsions resulting from heightened reflex irritability. The patient, as a rule, should have his strength conserved by being kept quietly in bed, and the fewest possible outsiders should be allowed in the room. Artificial heat must be employed in case of coldness of the surface or of the extremities, and general stimulants, such as alcohol in small doses, ether subcutaneously, ammonia by inhalation, and strychnin, preferably by hypodermic injection, should be used as indications arise. In connection with each poison later on special details of treatment will be given.

POSTMORTEM EXAMINATION.

The postmortem examination should be conducted as directed in the article on that subject on page 23 *et seq.* of Vol. I. A few special points only need be considered here.

In case of suspected poisoning it is often thought sufficient to examine the alimentary tract alone; this view, however, is entirely incorrect, and in all cases a thorough examination of all parts of the body should be made, both to determine the presence or absence of disease and to disclose the remote effects of the poison. It is only by making a complete investigation that entirely reliable results can be secured, and this is especially important in the event of a judicial inquiry, when the exclusion of death from disease of the kidneys, heart, brain, or other distant parts is often of great value. It is also frequently considered sufficient in cases of suspected poisoning to remove the stomach only for chemical analysis, but this likewise is a mistake; a part of the bowels, both kidneys, and the liver should in all cases also be removed for chemical investigation, and it is desirable in most instances to preserve the brain, spinal cord, heart, any urine that may be found in the bladder, and a quantity of the blood; if death from chloroform, ether, or other anesthetic is suspected, the lungs also should be removed for examination.

Each organ should be placed in a separate wide-mouthed glass jar, and it is seemingly almost unnecessary to add that these and everything else with which the organs come in contact should be scrupulously clean. Occasional mistakes, however, have arisen from neglect of this point. The writer is personally acquainted with a case in which the life of an unquestionably innocent man was placed in jeopardy by the discovery of arsenic in small amount in the remains of his wife, who died somewhat suddenly, although without the slightest symptoms of arsenical poisoning. It was subsequently demonstrated that the poison entered

the organs at the time of the postmortem examination by their being placed in a bowl carelessly supposed to have been clean, but which really had adhering to it a small amount of a highly arsenical embalming fluid.

The jars containing the different organs should be carefully sealed as soon as practicable after the parts have been placed in them. An excellent means of accomplishing this is to wrap each jar separately in a piece of firm paper, which should be held securely in place by narrow tape or stout twine passing over and around the jar several times. Melted sealing-wax should be dropped over the top and bottom of the package as well as at numerous points on the exposed edge of the wrapping-paper and at intervals on the tape or twine, and impressions made in the wax with a private seal, or, in case this is not at hand, with the seal of a bank or express company or with some rare coin. If the sealing is properly done, it is quite impossible to gain access to the jar without mutilating the package, and it affords a perfect safeguard, therefore, against secret tampering with the organs while they are being conveyed to the chemist or the pathologist for examination.

The evidences afforded by a postmortem examination in a case of suspected poisoning are important, but are rarely conclusive. With the exception of the corrosives, whose caustic effects on the mouth, throat, and stomach are often entirely characteristic, no poison produces appearances that are not practically identical with those that *may* be occasioned by disease or other natural causes. It was formerly believed, and among the uninformed it is still supposed, that rapid decomposition of the body and extensive discoloration are evidence of death from poisoning; this, however, has long since been thoroughly disproved, poisons having no specific action in accelerating putrefaction, but, on the contrary, some of them, like arsenic and mercury, at times appear to have a preserving influence. Extreme rigidity of the muscles after death is generally produced by strychnin, but equal rigidity may be occasioned by ordinary rigor mortis, and in exceptional cases it may persist as long from the latter cause as from strychnin; a dark fluid condition of the blood usually follows poisoning by belladonna, aconite, and many other neurotic poisons, but a similar state is produced by uremia and by certain infectious diseases; fulness of the vessels of the brain is usually seen as the results of opium-poisoning, but the same condition is occasioned by inflammation of the brain and many other cerebral diseases; the congestion of the spinal cord which generally follows death from strychnin is also observed after death from tetanus, hydrophobia, and spinal meningitis; fatty degeneration of the liver and other internal organs is often produced by phosphorus and arsenic, but it is also found as the result of age and of certain diseases, such as tuberculosis, puerperal eclampsia, and acute yellow atrophy; and redness of the stomach and bowels, which are the commonest of all the postmortem indications of irritant poisoning, are uniformly observed as the consequence of the gastritis and gastro-enteritis of disease.

Brouardel[1] records numerous instances of sudden death from ulcer-

[1] *Annales d'hygiène publique*, 1902, vol. xlvii., p. 12.

ation and perforation of the stomach and bowels, from rupture of the heart, gall-bladder, and Fallopian tubes, from intestinal strangulation and impacted gall-stones and feces, from unsuspected diabetes and Bright's disease, and from other causes of auto-intoxication which in a marked degree presented the symptoms of acute poisoning. Failure to excrete the toxic substances produced after an exceptionally hearty meal or excessive indulgence in alcohol may likewise, he states, cause death under conditions which might suggest poisoning. According to this authority, in from 25 to 30 per cent. of all cases of sudden death, no lesion can be discovered to which it can be referred.

Death may occur from poisoning and no postmortem evidences of it be discoverable, and many cases of this kind are on record. Even the appearances that are the most characteristic, and which are usually looked for with the greatest confidence, may be entirely wanting. Caspar[1] records a case of death from strychnin in which there was not only no unusual rigidity of the muscles, but no other evidence of death from the poison; and in the case of Lucy Heidelmeyer, which the writer investigated in 1887, the stomach and bowels showed not the slightest evidence of irritation, but, on the contrary, the mucous membrane was paler than usual, although she died of acute arsenical poisoning, as a legal inquiry clearly demonstrated.

The chief value of a postmortem examination in a case of suspected poisoning, aside from securing organs for chemical analysis, lies in determining whether such appearances are present as are usually produced by poison, or at least are compatible with it, and in disclosing the presence or absence of natural causes of death. In regard to the first of these points, it should be borne in mind, however, that the postmortem appearances produced by no poison are always the same, and sometimes, as before stated, even the most characteristic marks may be wholly absent; and concerning the second point it is scarcely necessary to state that death may occur from poison even though the person at the time was suffering from a serious or even necessarily fatal malady.

No postmortem investigation can be considered entirely complete without a careful microscopic examination by a competent pathologist of at least the heart, kidneys, and liver, and also, if possible, of the brain and spinal cord. When practicable, a bacteriologic examination of certain portions of the body, especially the liver, spleen, kidneys, lungs, and the heart's blood, should be made to determine the presence or absence of bacteria, which we know may sometimes be the cause of fatal symptoms similar to those produced by some poisons. A bacteriologic and microscopic examination of the bowels may also be useful in cases of suspected poisoning showing dysenteric symptoms. These extended tests and examinations, however, while always desirable from a scientific standpoint, and, in case no poison is found in the body by chemical analysis, highly important, are rarely if ever essential to the investigation of a case when poison is discovered in well-marked quantity.

[1] *Vierteljahrsschr. f. gericht. Med.*, July, 1874, p. 7.

CHEMICAL ANALYSIS.

Since it is only in exceptional instances that a case can be positively established as one of poisoning, either by the symptoms or the postmortem appearances, or even by both combined, a final decision in regard to the matter can generally be reached solely by the aid of a chemical analysis. It should not be understood, however, that in all instances a chemical analysis is indispensable to prove that death resulted from poison, or that, in event of an analysis, the finding of poison is essential, for outside circumstances may be of such a conclusive character, and, in occasional instances, the symptoms and postmortem appearances may be so characteristic, as to remove all doubt as to the nature of the case. In the well-known Palmer-Cook case[1] that occurred in England in 1855, Palmer was tried, convicted, and executed for killing Cook with strychnin, although chemical analysis failed to reveal the presence of the poison in the body after death; the incriminating circumstances were so strong, and the symptoms displayed by the deceased so characteristic, as to prove conclusively the guilt of the accused.[2]

It is commonly supposed that the stomach with its contents is the only organ necessary for chemical analysis in a case of suspected poisoning. While it is true that in most cases the stomach is the most desirable part of the body for examination, other organs should, whenever possible, also be submitted to analysis, and of these the most important, as a rule, are the liver, kidneys, and bowels, in the order in which they are named. The brain, spinal cord, heart, blood, and urine are frequently of service, and, in fact, there is scarcely a part of the body that may not be examined with profit. In the case of William Avery, who died of suspected poisoning at Fort Collins, Colorado, in 1890, the discovery of arsenic in the muscles, skin, and bones furnished important evidence after all the viscera and the brain had been removed and consumed in inconclusive tests. It should be remembered that the poison found in the stomach and that extracted from the other organs have a quite different relation. The poison in the stomach is not, as a rule, the toxic material that has produced death; it has not yet entered the circulation, and any damage that it may have done is purely local; on the other hand, the poison in the liver, kidneys, brain, etc., is the part that has caused the death of the person; it has been absorbed, has circulated in the blood, and has been deposited in the parts from which it is extracted. It should also be borne in mind that a person may die from the effects of a poison, and none be found

[1] For a full account of this celebrated case, see Tardieu, *Ann. d'hygiène*, 1856, vol. vi., p. 371 *et seq.*

[2] It should perhaps be stated that Palmer's guilt has not been universally admitted. Recently Brouardel (*Ann. d'hygiène*, 1902, vol. xlvii., p. 28), after reviewing the case, concludes that Cook died not of strychnin, but of uremic poisoning. The postmortem examination showed, he believes, that the deceased was suffering from undiscovered Bright's disease, and that excessive indulgence in alcohol and the table brought on a sudden attack which ended his life. Brouardel, however, evidently overlooks the moral circumstances, some of which were almost overwhelmingly convincing.

in the stomach, it having been entirely removed from that organ by vomiting and absorption. It is true that after death from poison none of the latter may be found by analysis in any part of the body, but it is more likely to have disappeared entirely from the stomach than from the other organs.

In addition to the various parts of the body, vomited matter, suspected food, any urine that may have been passed during life, medicines, and clothing may often be submitted to chemical examination with great profit, important light frequently being thrown on a case by the results of such an investigation. In the Maybrick case the discovery of arsenic in the handkerchief and in the clothing of the accused constituted one of the most important evidences against her.

The chemist who is called upon to make the analysis should be not only one who is thoroughly trained in general chemistry but also one who has a good knowledge of anatomy, physiology, materia medica, and toxicology. His integrity should be above the remotest suspicion. The suggestion[1] that two chemists should perform the analysis together, and should conjointly give testimony regarding the results, is an excellent one, and should be carried out whenever practicable. The sole objection to this plan is the greatly increased expense, but when life, liberty, and reputation are at stake this ought not to weigh.

The analysis should be conducted in a room whose doors and windows are kept locked and sealed at all times when the chemist is not personally present; no one except the analyst himself should be admitted to the room during the progress of the investigation, and under no circumstances should any poison, other than the reagents used in the analysis, be permitted in the apartment. All chemicals used in every operation should be known from actual tests by the chemist himself to be free from all impurity; the guaranty of dealers in chemicals should never by itself alone be depended upon. It is exceedingly desirable that coincident with the analysis of each part a blank test should be made in which an artificial mixture of foods, or a part of a calf's liver, equal in weight to that of the organ operated upon, is subjected to exactly the same procedure as is the part in question, the same chemicals in the same quantity being used in both. Unless the result of the blank test shows no poison the analysis of the suspected organ cannot be depended upon.

It is sometimes urged[2] that in every analysis for poison new apparatus should be used. In the opinion of the writer, however, this is not only not necessary, but is scarcely to be recommended. It is well known that articles of glassware, and sometimes of porcelain, occasionally contain arsenic and other poisonous metals which may be imparted to material placed in contact with them. For this reason it is far better to use apparatus which has previously been employed in a blank test that has given negative results, conclusive evidence being thus obtained that none of the vessels contain poisonous constituents, or at least yield none

[1] R. Ogden Doremus, *The Forum*, Oct., 1893.
[2] Dragendorff, *Ermittelung von Giften*, p. 14; Reese, *Manual of Toxicol.*, p. 243.

to the material under examination. It seems almost unnecessary to add that every piece of apparatus must be scrupulously clean. Each article should be washed separately just before using it, preferably first with dilute alcoholic solution of potassium hydroxid, then with warm dilute hydrochloric acid, and finally with an abundance of warm water.

As a rule some indication is afforded by the symptoms, postmortem appearances, or circumstances surrounding the case, of the poison that may be present, and this is often a most important guide in conducting the chemical analysis. This is especially true in those numerous cases in which, on account of expense or lack of sufficient material, a direct test must be made for one poison or group of poisons. In all instances, however, where it is practicable a complete search should be made, so as not only to determine fully what poison or poisons are present but also to afford, what in many cases is of nearly equal value, evidence of the absence of other toxic substances.

Previous to beginning the chemical examination of any organ it should be thoroughly inspected and weighed; and in case of the contents of the stomach, vomited matter, blood, or other fluid or semifluid substances, the material should also be measured. The interior of the stomach and the stomach-contents should be most critically examined, both with the unaided eye and with a hand magnifying-glass, for the presence of undissolved poisons, such as arsenic trioxid, Paris green, lumps of opium, and the seeds, leaves, and other parts of poisonous plants, and also for mechanical irritants, like powdered glass. The evidence thus afforded is frequently of the most convincing character. The odor of the material should also be carefully determined and the reaction to litmus-paper observed. In case of the contents of the stomach or of vomited matter, the closest attention should be paid to the nature of the food present, a microscope of medium power often being used with great advantage to demonstrate the presence or absence of starch granules, muscular fiber, and vegetable tissue, and to disclose their exact character. The identity of the material under examination and very many other points are not infrequently established by the evidence thus obtained.

The first step in a chemical analysis for poisons is usually the separation of the latter from the accompanying organic material, and the processes in general for accomplishing this are given below. It is only after such separation, and subsequent purification if necessary, that chemical and physiologic tests can ordinarily be applied with any degree of certainty, the presence of complex organic matter generally being fatal to the development of conclusive reactions for most poisons. In connection with each individual poison the characteristic tests for it will be given, and also the methods which may be employed for extracting it from the various organs; we give below, however, a systematic procedure for separating poisons in general, and while in many cases this scheme may advantageously be simplified or modified in many ways, the general principles involved are such as are most usefully employed in nearly all instances.

The substance to be analyzed should first be very finely comminuted

with a pair of sharp scissors, chopping-knife, or hashing machine, or by pulverizing in a mortar, and it is then divided into three equal portions, one to be set aside for use in case of accident or for additional tests if any are needed; another to be used for the extraction of volatile and subsequently of mineral poisons; and the third to be employed for the detection of organic poisons.

In some cases when the amount of material is very small, or the quantity of poison present is believed to be minute, it may be necessary to divide into two parts only, one for the volatile and mineral poisons, and the other for the organic poisons, no portion being set aside as a reserve. So, too, if circumstances point to the administration of a corrosive acid or alkali, it is necessary, if a complete examination is to be made, either to divide the material into four parts (one being used for testing for these substances and the others disposed of as above), or to devote the third reserved portion to this purpose. It is obviously,

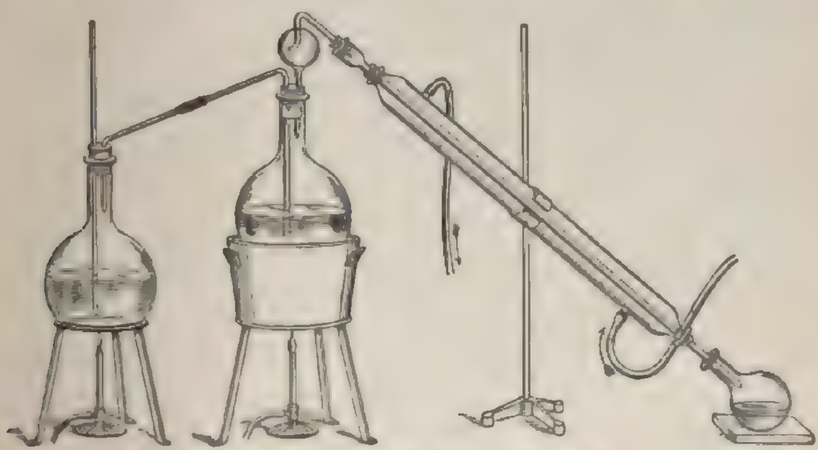


FIG. 24.—Apparatus for distilling in steam.

however, very important whenever practicable to keep some part of each organ or substance tested as a reserve supply. Accidents may happen even with the most careful manipulation, or additional and unexpected tests may be needed as the investigation proceeds, and the reserved part may become almost indispensable.

If the process is properly conducted, the same portion of material may be used without loss or other inaccuracy for the separation of volatile poisons and then for the detection of mineral substances.

Separation of Volatile Poisons.—The material brought to the consistence of a thin gruel by the addition of distilled water if necessary, and made of slightly acid reaction (if not already so) by adding a little tartaric acid, is placed in an apparatus for distilling in steam, which is connected with a Liebig condenser through which very cold water circulates (see Fig. 24). Heat is carefully applied and the distillate is collected in several fractions, each one amounting in volume to about

one-fifth of the material in the flask. The operation should be conducted in a dark room, and with a screen placed between the flame of the lamp and the condenser, in order that any phosphorescence, indicative of the presence of phosphorus in the material under examination, may be seen.

After three or four fractions have been collected, or sooner if it appears that nothing further is distilling over, the apparatus is allowed to cool and magnesia carefully added until the mixture is of very feebly alkaline reaction. The mixture is then again distilled as before.

The *distillate from the acid mixture* may contain (among other less important volatile poisons) alcohols, aldehyds, ethers, carbolic acid and other phenols, bromin, iodin, phosphorus, camphor, chloral, chloroform, turpentine and other volatile hydrocarbons, essential oils, nitroglycerin, nitrobenzene, benzoic acid, salicylic acid, hydrocyanic acid, hydrochloric acid, and other volatile acids.

The *distillate from the alkaline mixture* may contain ammonia, anilin, coniïn, nicotin, and other volatile bases.

To each of these distillates the appropriate tests for the several poisons that may be present are to be applied as directed in subsequent chapters in connection with the individual poisons.

A number of the substances in the above lists, such as camphor, salicylic acid, anilin, coniïn, nicotin, and other volatile bases, may appear also in the results of the examination for organic poisons, as will be seen later.

After the two distillations as described above are completed, the contents of the flask are washed into a porcelain capsule, any excess of moisture from condensed steam removed by careful evaporation on the water-bath if deemed necessary, and the mixture then submitted to examination for the detection of inorganic or mineral poisons, nothing having been done in the above operations to destroy or volatilize them.

Examination for Mineral Poisons.—A number of processes have been devised for the extraction of mineral poisons in toxicologic investigations, in almost all of which the first procedure is the destruction, more or less complete, of the organic matter with which the poison is associated. One of the earliest methods suggested for this purpose was the actual combustion of the organic matter, either alone or after mixture with certain oxidizing agents like potassium nitrate, as practised by Orfila,[1] the father of modern toxicology. In these processes the material to be operated upon is properly dried, introduced into a crucible or other suitable vessel, and heated in contact with the air until the organic matter is completely burned away; the ash is then treated with acidulated water and the solution obtained tested for the presence of poison. This process has the advantage of simplicity and directness, and is still sometimes used, as, for instance, occasionally in testing urine for the presence of lead. Since, however, during the combustion a number of the most important mineral poisons, such as arsenic and

[1] Orfila, *Traité des poisons, ou toxicologie générale*, 1818, second edition, vol. i., pp. 105, 207, etc.

mercury, are partially or wholly expelled and lost, the method has but limited application. The most generally useful processes are those in which chemical reagents are employed to remove the organic matter, and there are two chief methods at present in use: the first, that of Gautier, in which nitric and sulphuric acids are the destroying agents; and the second, that of Fresenius and Babo, in which the oxidizing effect of potassium chlorate when heated with hydrochloric acid is taken advantage of.

Process of Gautier.[1]—This process, founded on the older methods of Danger and Flandin[2] (in which sulphuric acid alone was used) and of Fihol[3] (in which a mixture of nitric and sulphuric acids was employed), has itself been modified more or less by Chittenden and Donaldson[4] and by Chapuis.[5] The method is best carried out as follows:

One hundred grams of the material to be examined are placed in a capacious casserole and treated with 30 grams of nitric acid and moderately heated. The mixture liquefies and assumes an orange hue, when it is removed from the heat and 6 grams of sulphuric acid added. This usually causes a somewhat violent reaction and the mass becomes dark brown. It is again heated, either on a sand-bath or air-bath, until carbonization begins and the black mass commences to adhere to the side of the vessel; it is then removed from the source of heat, allowed to cool, and 10 or 15 grams of nitric acid distributed over the mass drop by drop. It is again heated until white vapors have completely disappeared, when the mass is allowed to cool, pulverized, and boiled with two or three successive portions of water containing a sixth of its volume of hydrochloric acid. The mixture is filtered hot; in the carbonaceous mass left on the filter are to be found, in sparingly soluble combination, more or less of the silver, lead, bismuth, tin, barium, and strontium, which may have been present in the original material, while in the filtrate are all the other metals, excepting mercury, which in this process is either entirely or in great part lost by volatilization. The residue and the solution, being now free from organic matter, may be examined by the usual systematic procedures of qualitative analysis, if a complete search for all metals is to be made; or if but a single substance, such as arsenic, antimony, or lead, is sought, more direct and frequently more delicate tests may be used, such as are described in connection with the individual poisons in the succeeding section.

The process as described above and the various modifications alluded to have, however, at least one serious inconvenience—the conversion of the organic material in the final stage into a solid carbonaceous mass from which the complete extraction of the metallic poison is extremely difficult. It is true that by pulverizing the material as directed and repeatedly treating it with hot acidulated water, most of the mineral substances are almost entirely dissolved out. Traces, however, of even the more readily soluble are likely to be lost, and, as already stated,

[1] *Annales de chimie et de physique*, 1876, fifth series, vol. viii., p. 384.

[2] Danger et Flandin, *De l'arsenic*, Paris, 1841, p. 35; Flandin, *Traité des poisons*, vol. i., p. 618.

[3] *Thèses de la Faculté des Sciences de Paris*, 1848.

[4] *American Chemical Journal*, vol. ii., p. 235.

[5] *Précis de toxicologie*, p. 150.

silver, lead, bismuth, tin, barium, and strontium remain to a greater or less extent in the carbonaceous residue, and can be tested for only with some difficulty. To remedy these defects Gautier[1] has more recently modified his method, using a larger amount of nitric acid and a lower degree of heat, in such a manner as to give, after the destruction of the organic matter, not a solid carbonaceous mass, but a liquid material from which the extraction of the various metallic poisons in their full amount is easy. This modification, like the original process, was devised especially for the detection of arsenic, and for this purpose it is particularly useful, but it may be advantageously employed for the separation of any of the metals except mercury, a part or even all of which may be lost, as in the original method.

Gautier's modified process, as practised by the writer, is as follows: On 100 grams of the material there are poured from 30 to 60 grams of pure nitric acid, the amount depending upon the character of the material operated on. One gram of sulphuric acid is now added and the mixture is heated, preferably on a water-bath at first, in a porcelain capsule until liquefaction is produced and the material becomes thick. It is then removed from the heat and not more than 8 or 10 grams of pure sulphuric acid are added. The mixture is heated again rather strongly (care being taken, however, here and elsewhere, that the temperature shall not be high enough to produce carbonization), taken from the heat, and nitric acid poured over the material a little at a time until upon heating it to a point where thick vapors of sulphuric acid are given off there is left in the capsule a brown liquid practically uncarbonizable at the temperature at which sulphuric acid commences to boil. Usually about 50 grams of nitric acid are required for this purpose, but sometimes a considerably larger amount is needed. In certain cases the destruction is difficult, and successive portions of nitric acid have to be added many times. When a point is finally reached where the nitric acid produces scarcely any further oxidizing effect, the acid is once more driven off by heat, the material allowed to cool, a little sulphuric acid added, and the small quantity of residual brown liquid poured with constant stirring into 600 to 700 c.c. of distilled water. The capsule is washed, and the washings are added to the previous liquid. A finely subdivided brownish material falls to the bottom of the receptacle, and the supernatant liquid is more or less dark in color. The liquid is filtered. The residue on the filter is to be tested, as in the original process, for silver, lead, bismuth, tin, barium, and strontium, while the filtrate is examined for traces of these (which may have passed more or less into solution) and for all the other metals.

Process of Fresenius and Babo.[2]—This is much the most generally useful of the various methods that have been devised for the destruction of organic matter; it enables one to test for all mineral poisons, none being volatilized as in the process of Gautier. The procedure is based on the fact that when potassium chlorate and hydrochloric acid are heated together, chlorin and oxids of chlorin are abundantly evolved, which

[1] *Comptes rendus*, 1899, vol. cxxix., p. 936.
[2] *Annal. d. Chemie u. Pharmacie*, 1844, vol. xlix., p. 308.

have an energetically destructive action on almost all forms of organic material. The process is founded on the older method of Jacquelain,[1] in which a stream of chlorin conducted into the mixture was used for the same purpose.

In using the process, the suspected material is placed in a capacious evaporating dish or flask, mixed with about a third of its volume of hydrochloric acid, and gently heated on a water-bath; from time to time pinches of pulverized potassium chlorate are added, and the volume of the mixture should be kept up by occasionally pouring in a little distilled water. The addition of the chlorate is continued until the mixture has a thin consistence, a uniform straw-yellow color, and no particles of organic matter remain suspended in the liquid excepting more or less fat, which in this process is only incompletely attacked. The mixture is now filtered and the insoluble material thoroughly washed with hot water. In the residue on the filter are to be found any silver that may have been present, in the form of the chlorid, and more or less of the lead, barium, and strontium, as sulphates, and these metals are to be tested for in this mixture. The amount of lead, barium, and strontium found in the residue depends on the quantity of sulphuric acid or sulphates present in the original substance or produced by oxidation from the sulphur of the organic compounds during the destruction of the latter. Barium and strontium are likely to be completely precipitated, but lead, unless in large amount, may pass entirely into solution, its sulphate being somewhat soluble in excess of warm hydrochloric acid, especially in the presence of chlorids of sodium and potassium.

The filtrate, in which are to be found all the other metals, is to be moderately heated, and any excess of chlorin removed by passing through it a rapid stream of carbon dioxid. Sodium sulphite is then added in sufficient quantity to cause the liquid to smell strong of sulphur dioxid, and the gentle heating continued until the odor of the dioxid has disappeared. The object of using the sulphite (for which sulphurous acid may be substituted if more convenient) is to reduce any arsenic present from the form of arsenic acid, in which it exists in the liquid, to arsenous acid, in which state it is much more easily precipitated by hydrogen sulphid in the succeeding step of the process. If the liquid is exceedingly acid, it is well to neutralize it partially by the addition of sodium carbonate (for in the presence of too great acidity antimony and tin are likely to be imperfectly or even not at all precipitated by hydrogen sulphid), after which it is put in a flask, the temperature raised to and kept at about 70° C. (158° F.), and a washed stream of pure hydrogen sulphid from an automatic generator (see Fig. 25) passed somewhat slowly through it for three or four hours. It is then allowed gradually to cool, the stream of hydrogen sulphid still continuing, and when entirely cold the gas is stopped, the flask well stoppered, and set aside in a warm place. At the end of twenty-four hours it is again heated to 70° C., treated with hydrogen sulphid exactly as before, the flask again stoppered, and once more set aside in a warm place for another twenty-four hours. If at the end of this time the liquid

[1] *Comptes rendus*, 1843, vol. xvi., p. 28.

smells strong of hydrogen sulphid, as is almost always the case if the operation has been properly conducted, the precipitation may be considered complete; but if there is little or no odor of the gas, the operation as directed above must be repeated until the desired result is obtained. The contents of the flask are then thrown on a filter, and the precipitate well washed with water containing a little hydrogen sulphid in solution. The precipitate contains in the form of sulphids any mercury, bismuth, cadmium, copper, arsenic, antimony, tin, gold, and platinum, and a part or all of the lead, that may have been present in the original material, while in the filtrate are all the other metals excepting silver,

FIG. 25.—Apparatus for hydrogen sulphid.

barium, and strontium, which, as we have seen, are left in the residue after the destruction of organic matter by potassium chlorate. The precipitated sulphids and the filtrate may be examined by the usual methods of qualitative analysis; or if only one or two metals are in question, shorter and perhaps more delicate tests may be used, as suggested in connection with the Gautier process.

Process of Ogier.[1]—Two objections have been urged to the process of Fresenius and Babo, namely, that small quantities of arsenic may be carried off as the volatile chlorid and lost, and that in projecting the chlorate into the acid mixture a considerable portion of the chlorin is

[1] *Le laboratoire de toxicologie*, p. 41.

evolved on the surface of the fluid and passes away without effecting destruction of any of the organic matter, necessitating the use of an excessive amount both of the chlorate and of the hydrochloric acid. To overcome these defects, Ogier has modified the method of conducting the process by placing the material to be operated on in a flask, mixing it with the estimated amount of chlorate required for complete destruction of the organic matter (usually 1 part of the chlorate to 8 or 10 of the solids to be destroyed), and passing into the mixture a stream of hydrochloric acid gas liberated from the aqueous acid by allowing sulphuric acid slowly to drop into it. The operation is carried out in an apparatus shown in Fig. 26, in which *a* is the flask in which the organic mixture and chlorate are placed, *b* the generator for producing the hydrochloric acid gas, *d* a bottle for washing the gas, and *f* a vessel in which the fumes given off from *a* are condensed so as to

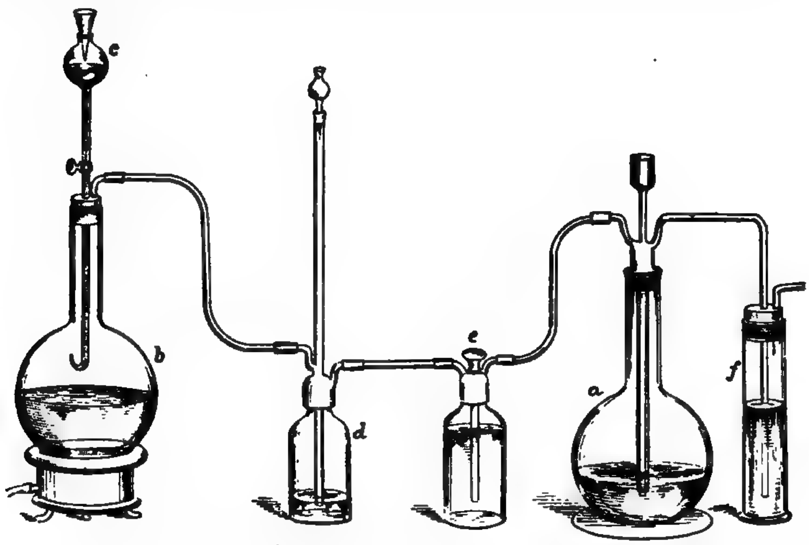


FIG. 26.—Apparatus for the destruction of organic matter (after Ogier).

avoid all loss of arsenic. *e* is a bottle provided with a three-way stopcock, so that in case the gas comes over too freely it may be diverted into the bottle and absorbed by the water in it. As the decomposition of the chlorate takes place in this process not on the surface of the mixture, but all through the mass, the organic matter, it is claimed, is more thoroughly and quickly destroyed and with a less expenditure of chemicals than in the original method; and as the operation is conducted in a closed vessel connected with a condenser, there should be no loss of arsenic. There are some inconveniences, however, connected with the process; it requires considerable care to keep the numerous parts of the apparatus perfectly clean, and, what is even more serious, when the hydrochloric acid gas comes in contact with the warm chlorate mixture, the chemical reaction under some conditions may become so vigorous as to fracture the flask and cause loss of material unless the process is conducted with constant attention. In spite of these drawbacks the

method is to be recommended, especially when the quantity of poison present is believed to be small, since the amount of chemicals introduced in destroying the organic matter is reduced to a minimum and there is no loss of material.

Process of Reinsch.[1]—In the processes given above for the extraction of mineral poisons the organic matter is destroyed as the first step; a very excellent method, however, was devised by Reinsch, in 1841, for the detection of certain of the poisonous metals without the previous destruction of organic substances. The process is one of great simplicity, and, if properly conducted, of thorough accuracy and considerable delicacy; it is applicable, however, to the detection of arsenic, mercury, and antimony only, but for rapid work, and especially for clinical use, it is of the greatest value. It is a qualitative test only, although an idea may be gained in a general way of the relative quantity of the poison present by the rapidity and intensity of the reactions obtained.

The process may advantageously be carried out as follows: The suspected material properly comminuted is put in a beaker, flask, or porcelain capsule, mixed with a quarter of its bulk of pure hydrochloric acid, water added, if necessary, to bring it to a thin consistence, and the mixture boiled for five or ten minutes, or until the substance under examination is thoroughly disintegrated. A piece of pure copper foil about half an inch long and quarter of an inch wide, well polished by rubbing with fine emery paper, is now dropped in the mixture and the boiling continued a few minutes, the piece of foil being taken out at the end of each minute and inspected. For this purpose it is very convenient to hang the foil on a piece of platinum wire, but this is by no means essential. If at the end of ten minutes the foil has received no deposit, we may be sure that the above poisonous metals are either not present at all, or, at most, in only very minute proportion; but, on the other hand, if the foil is coated, it does not necessarily indicate that one of these substances is present, for certain other metals—gold, silver, platinum, palladium, tin, and, most important of all, bismuth—may also be deposited on the foil in an analogous manner.

If a deposit is obtained, to determine whether one of the poisonous metals has produced it, several pieces of copper foil are treated in the same manner as the first, and when all have been coated, they are washed with water, then with alcohol, and finally with ether, after which they are dried, placed in a small test-tube or sublimation-tube, the diameter of which should not exceed a quarter of an inch, and gently heated in a small flame. If arsenic, mercury, or antimony is present, a sublimate will be produced in the colder part of the tube, and by examining this under the microscope, its nature can be determined, arsenic appearing in the form of octahedral crystals (see Fig. 37, p. 416), mercury as metallic globules (see Fig. 36, p. 396), and antimony either in the form of amorphous granules or as needle-shaped crystals. Occasionally antimony produces a sublimate of octahedral crystals somewhat resembling those of arsenic,[2] but distinguishable from the latter by the fact that they

[1] *Jour. fur praktische Chemie*, 1841, vol. xxiv., p. 244.
[2] Wormley, *Amer. Jour. Med. Sci.*, Oct., 1877, p. 399.

are volatilized by the application of a high degree of heat only (between 700° and 800° C.), while those of arsenic are sublimed at a comparatively low temperature (about 200° C.). Neither bismuth nor any of the other metals mentioned above produces any sublimate, and they can be distinguished as a class, therefore, from the poisonous metals very easily.

It is highly important that the subliming-tube be thoroughly clean and dry, and the foil free from all grease. Failure to observe these conditions may produce confusing and uncertain results. In manipulating the foil after it has been washed with ether it should preferably not be touched with the finger, but handled with forceps, to avoid imparting any trace of oil to it.

Sometimes, in applying the test, the copper foil becomes more or less stained by the action of the sulphur and possibly of some other normal constituents of the organic matter operated on; but upon heating the foil thus coated in the subliming-tube, either no sublimate is obtained, or an amorphous one, which can very easily be distinguished under the microscope from the sublimates produced by the poisonous metals.

It should be added that sulphur in certain inorganic compounds, such as sulphids, sulphurous acid, and thiosulphates, also produces a deposit on the foil, and the same is true of the compounds of selenium and tellurium. These bodies, however, especially the two last, are rarely present in mixtures submitted to the test, and, moreover, they may be readily differentiated from the poisonous metals by their behavior when the coated foil is heated in a tube. All three under this treatment give slight sublimates which condense but a short distance from the foil; that from sulphur is yellowish and either amorphous or in acicular crystals, and those from selenium and tellurium appear in colorless needle-shaped or cubical crystals which arrange themselves in characteristic fern-like masses.

It is very desirable, whenever possible, to confirm the character of the sublimate by other tests, and this can be accomplished easily as follows: A small crystal of iodin is placed in the tube, which is then sealed with wax and put aside in a warm place for several hours, when, if mercury is present, it will be converted into mercuric iodid and the sublimate will assume the bright-red color of that compound. If the result of this test is negative, the iodin is removed, the portion of the tube containing the sublimate is cut off with a file and placed in a test-tube; distilled water is added and boiled, by which arsenous oxid, if present, is dissolved out, and may be tested for in the solution by the action of hydrogen sulphid or of other liquid tests for arsenic. If the test for arsenic is also negative, the piece of glass tubing containing the sublimate should be boiled with a solution of tartaric acid, which dissolves oxid of antimony, and its presence in the liquid may be demonstrated by hydrogen sulphid or by other tests for antimony. It should not be forgotten, however, that more than one of the metals may be present, in which case we get, both in the appearance of the sublimate under the microscope and in the subsequent identifying reactions, results that characterize the different metals present.

A very important point in connection with the test is that the production of a coating on the copper foil must never be taken as proof of the presence in the mixture under examination of a poisonous metal; subsequent sublimation, and the examination of the sublimate under the microscope, must invariably be practised before any conclusion should be drawn.

Detection of Organic Poisons.—Laying aside volatile organic poisons, which have already been considered, organic compounds of toxicologic interest may with few exceptions be divided into four groups, as follows: (1) alkaloids and other basic substances; (2) glucosids and other neutral principles; (3) acids; (4) oils, gums, resins.

Of the above, the most important from a toxicologic standpoint are the alkaloids, and the earliest systematic processes for the detection of organic poisons were devised for their extraction; by a modification, however, of these processes most or all bodies belonging to the other classes may also be detected, if discoverable at all. Stas, of Belgium, was the first to introduce a satisfactory method for the extraction of alkaloids for toxicologic purposes, the process having been invented for the detection of nicotin in the body of M. Fougnies, who was poisoned with that alkaloid in 1850 by his brother-in-law, Count Bocarmé. His method, which may be said to be the origin of all general processes for the detection of organic poisons, is, in brief, as follows:

Process of Stas.[1]—The finely divided material is mixed with two or three times its volume of strong alcohol acidulated with tartaric or oxalic acid, gently heated,—the temperature never exceeding 75° C. (168° F.),—filtered when cold, and the filtrate evaporated in a vacuum or in a current of air, at a temperature not above 35° C. (95° F.). If any insoluble matter separates during evaporation, the liquid is again filtered, evaporated still further in a vacuum, the residue extracted with absolute alcohol, and the solution thus obtained evaporated at a low temperature. The residue is taken up with a small quantity of water, filtered, the filtrate rendered alkaline with sodium bicarbonate and agitated at once with several times its volume of ether; the latter is drawn off and allowed to evaporate spontaneously, when any alkaloid which may have been present in the substance under examination is left behind.

Numerous modifications of Stas' method have been introduced.

Otto[2] suggested agitating the final acid liquid before adding sodium bicarbonate with ether, which removes coloring-matter, glucosids, and many other non-alkaloidal substances, which may be recovered and tested upon evaporating the ether. This modification, commonly known as the *Stas-Otto process*, is greatly to be preferred to the original as furnishing the alkaloid in a much purer state.

Rodgers and Girdwood[3] extracted with dilute hydrochloric acid, and suggested the use of chloroform instead of ether. While their process was devised particularly for the isolation of strychnin, it is applicable for the detection of most of the alkaloids, and has the advantage over

[1] *Bulletin de l'Acad. de Méd. de Belgique*, 1851, vol. xi., p. 304.
[2] *Annal. d. Chem. u. Pharm.*, 1856, vol. c., p. 44.
[3] *Lancet*, June 28, 1856, p. 718; *Pharm. Jour. and Trans.*, vol. xvi., p. 497.

the original Stas method, that chloroform is a much better solvent of most alkaloids than ether is, a fact that has justly caused it largely to supplant the latter in general alkaloidal extraction.

Uslar and Erdmann[1] also extracted the material under examination with water slightly acidified with hydrochloric acid, alkalinized the filtered liquid with ammonium hydroxid, and shook out with hot amyl alcohol, in which alkaloids are freely soluble, and which has the advantage over ether and chloroform of readily dissolving out morphin, which is nearly insoluble in the other two liquids.

All the above and a number of other modifications of the Stas process are founded on the following general principles: (*a*) Water or alcohol, acidulated with acetic, tartaric, hydrochloric, or other acid, extracts from complex mixtures any alkaloids present in the form of readily soluble salts, along with glucosids and most other organic bodies of toxicologic interest, together with many impurities. Ligneous fibers, insoluble proteids, starch, fats, resins, and many other similar substances are wholly or chiefly left undissolved and are separated by filtration. (*b*) If the solution thus obtained after expelling alcohol (if any has been used) is agitated with ether, chloroform, or other analogous immiscible liquids, glucosids, acids, etc., are dissolved out, and may be recovered for examination by evaporation of the immiscible solvent, while the alkaloidal salts are left behind in the acid aqueous solution. (*c*) If the latter is rendered alkaline by ammonium hydroxid, potassium hydroxid, sodium carbonate, or other similar body, any alkaloidal salts present are decomposed, the acid being abstracted, and the free alkaloids, which, as a rule, are sparingly soluble in water, are thrown out of solution. (*d*) If this mixture is now agitated with ether, chloroform, or other similar immiscible fluid, any alkaloids present pass almost wholly into solution in the immiscible solvent, and if the latter is drawn off and evaporated, the alkaloids are left behind in the residue, and may be identified by appropriate tests, particularly after purification.

Process of Dragendorff.[2]—Following these general principles, and in addition taking advantage of the different solvent powers of various immiscible liquids upon alkaloids and other organic bodies when in acid and in alkaline solutions, Dragendorff has devised an elaborate and valuable process not only for the extraction of nearly all non-volatile organic compounds of toxicologic interest, but also for their separation into a number of groups to facilitate their identification. In cases of suspected poisoning in which a complete examination is to be made, and particularly in those in which no clue is afforded to the poison present, this method or one of its modifications is of the greatest utility. It is, however, somewhat long and complicated, and when only one or two organic substances are to be looked for it may very advantageously be materially changed and much more direct methods employed, such as are given later on in connection with the individual poisons.

The process is carried out as follows: The finely divided material is digested for a number of hours with water acidulated with sulphuric

[1] *Annal. d. Chem. u. Pharm.*, 1861, vol. cxx., p. 121.
[2] *Ermittelung von Giften*, 1895, p. 149.

acid at a temperature of 40° C. The solid material is pressed and again extracted with dilute acid and pressed as before. The united fluids obtained by expression are filtered and the filtrate evaporated at a gentle heat to a syrupy consistence, when it is mixed with several times its volume of alcohol to precipitate proteids, the mixture digested for several hours at a moderate heat, allowed to cool, and filtered. The filtrate is freed from alcohol by gentle heat, the aqueous residue diluted with water if necessary, and clarified by filtration. The acid liquid thus obtained is shaken out with petroleum ether in several successive portions until nothing further is removed by it, as is shown by leaving no residue upon evaporation. The liquid while still acid is then similarly extracted first with benzene and then with chloroform, after which the fluid is shaken with petroleum ether in order to remove traces of chloroform, and ammonia is added to alkaline reaction.

The alkaline liquid is now extracted successively with petroleum ether, benzene, chloroform, and amyl-alcohol, after which the fluid is evaporated to dryness with the addition of powdered glass, and the finely divided residue is extracted with chloroform.

By evaporating the several immiscible solvents used above, residues are obtained containing the various substances extracted by them, which, according to Dragendorff, are chiefly as follows:

Petroleum Ether from the Acid Solution.—Piperin, picric acid, salicylic acid, benzoic acid, camphor and ethereal oils, capsicin, and the esters of salicylic and benzoic acids with guaiacol, naphthol, and cresol.

Benzene from the Acid Solution.—Caffein, geissospermin, traces of veratrin and hydrastin, piperin, cantharidin, anemonin, santonin, caryophyllin, cascarillin, cubebin, aloëtin, elaterin, colocynthin, populin, digitalin, strophanthin, picric, benzoic, and salicylic acids, traces of hydroquinon and resorcin, and salophen.

Chloroform from the Acid Solution.—Theobromin, colchicin, papaverin, narcein, hydrastin, cinchonin and cinchonidin, jervin, aspidospermin and quebrachin, antifebrin, picrotoxin, colocynthin, esculin, and gelsemic acid.

Petroleum Ether from the Alkaline Solution.—Coniïn, nicotin, spartein, lobelin, pyridin, anilin, kairin, thallin, phenocoll, antipyrin, aconitin, delphinin, and traces of strychnin, brucin, gelsemin, emetin, quinin, veratrin, and the alkaloids of quebracho and of geissospermum.

Benzene from the Alkaline Solution.—Strychnin, brucin, gelsemin, emetin, quinin, veratrin, and the alkaloids of quebracho and geissospermum (traces of which, as shown above, have already been removed by petroleum ether from the alkaline solution); cocain, atropin, hyoscyamin, hyoscin, physostigmin, eserin, pilocarpin, narcotin, codein, thebain, apomorphin, antipyrin, and thallin.

Chloroform from the Alkaline Solution.—Berberin, what is left of the cinchona alkaloids, of papaverin, and of narcein, and slight traces of morphin.

Amyl-alcohol from the Alkaline Solution.—Morphin, solanin, salicin, and traces that may have been left of certain other bodies, such as narcein, cystin, saponin, etc.

Curarin is not removed by any of the above solvents, but is extracted from the residue left after evaporating the fluid with powdered glass by treatment with chloroform.

Each of the above residues should be examined for the various substances which may be present in it by identifying tests such as are fully described in connection with the different poisons. The residues are, however, rarely pure enough to be tested at once, but generally require purification by solution in acidulated water and reëxtraction by the appropriate immiscible solvents; and this process of purification sometimes has to be repeated a number of times.

In this and in all other processes in which shaking out with immiscible solvents is practised, emulsions of the latter with the aqueous liquid not infrequently are produced and greatly embarrass the procedure. From these emulsions the immiscible solvent separates with great tardiness, hours and even days sometimes passing before the separation is complete. To overcome such emulsions many suggestions have been offered, such as adding an additional amount of the solvent, gently heating the mixture in a water-bath, slowly stirring with a glass rod, and submitting to the action of a centrifuge. While one or all of these devices may often be used with advantage, the most satisfactory method is, as far as possible, to prevent the formation of the emulsion, and this can usually be accomplished by care in agitating the mixture. If, upon adding the immiscible solvent and shaking gently, it is seen that an emulsion is likely to follow, the agitation should be discontinued at once, the solvent allowed to separate, and after it has been drawn off a fresh portion poured in and again gently shaken. After the first or second extraction there is less likelihood of a persistent emulsion forming, although this is not invariably the case. In thus using gentle agitation only it is necessary to depend on repeated portions of the immiscible solvent to make a complete extraction; but the saving in time and patience, as well as the greater accuracy of the results obtained, much more than compensates for this slight disadvantage.

FIG. 27.—Separating funnel.

In this, as in all other processes of shaking out with immiscible solvents, a separating funnel (see Fig. 27) is of particular value, but a test-tube or flask may be used and the two layers of liquid separated from each other by the convenient device introduced by Prescott[1] (see Fig. 28). As will be seen by referring to the figure, this consists of a doubly perforated stopper provided with two glass tubes, one of which passes only just through the stopper, while the other is much longer and may be drawn up and down to suit different levels of the liquids.

[1] *Organic Analysis*, p. 36.

The method of using it will be evident from an inspection of the illustration.

The Author's Modification of the Dragendorff Process.—Dragendorff's process has two serious drawbacks: First, the use of so strong and disintegrating an acid as sulphuric in extracting the alkaloids from the tissues causes much foreign matter to go into solution and produces many new soluble compounds which still further contaminate the resulting extract, greatly to the disadvantage of the subsequent procedures. Second, on account of the presence of these many impurities the volume of the fluid when subjected to the shaking-out process is usually so considerable that the treatment with immiscible solvents requires the use of a large amount of the latter, and this impairs greatly the sharpness of division into groups, which is one of the chief merits claimed for the method.

In order to overcome these objections the author has modified the process as described below. As will be noted, sulphuric acid is replaced by tartaric or other vegetable acid, the first extraction is made with alcohol instead of with water, and subsequently the extract is purified repeatedly by treatment with an excess of strong alcohol. By these procedures the final fluid for shaking out is freed entirely from all proteid material and many other impurities, and is reduced to a comparatively small volume. When the immiscible solvents are applied, much less time and material are necessary, emulsions (such as embarrass the original Dragendorff process greatly) rarely occur, and the extracted alkaloids and glucosids are of far greater purity and their division into groups much sharper.

In conducting the process the material under examination is finely divided, placed in a capacious flask, and treated with three times its volume of 50 per cent. alcohol acidulated with acetic or tartaric acid. The mixture is heated on a water-bath to a temperature not exceeding 50° C. (122° F.) for an hour or two, and then filtered, the insoluble residue being thoroughly washed with dilute alcohol, strongly pressed, again digested with acidulated spirit, and the liquid filtered and residue pressed as before, the operation being repeated a number of times until thorough extraction is accomplished. The united filtrates and washings are evaporated at a moderate temperature, preferably not exceeding 60° C. (140° F.), to the consistence of a syrup, and, while it is still warm, three or four volumes of 90 per cent. alcohol are very slowly stirred in. The mixture is allowed to digest in a warm place with frequent agitation for at least an hour, when the liquid is filtered from the insoluble residue and the latter washed repeatedly with warm alcohol. The filtrate and washings are again evaporated to a syrupy consistence, as before described, and when cold taken up by the very gradual addition, with constant stirring, of three or four volumes of absolute alcohol; the mixture is filtered, and the insoluble residue thoroughly washed with absolute alcohol. The filtrate should now be tested by adding to a small portion of it in a test-tube an equal quantity of absolute alcohol; if a precipitate is produced, it is evidence that proteids and other coagulable

foreign matter have not been completely removed, and the liquid must be evaporated to a small bulk as before and again treated with a large excess of absolute alcohol; and this process must be repeated, if necessary, until all coagulable material has been eliminated. But if the alcohol occasions no deposit, the filtrate and washings are reduced by evaporation to a syrupy liquid, which when cold is taken up with two or three volumes of water containing a few drops of sulphuric acid. The mixture is filtered, the insoluble residue well washed with water, and the united filtrate and washings shaken out with repeated portions of petroleum ether until nothing further is extracted.

If a complete examination is to be made, the aqueous liquid is now agitated in turn with the various other immiscible solvents of the Dragendorff process. For this treatment it is well fitted, since, as before stated, all proteids and a large number of other foreign matters have been separated in the preparatory operation. It should not be large in bulk, and should not emulsionize easily upon being shaken out with the different immiscible solvents, if the various steps in the above process have been carefully carried out.

In case only one or two specific poisons are to be looked for, and a general examination for alkaloids is unnecessary, the acid liquid after being purified by treatment with petroleum ether may often with advantage be tested directly for the suspected substances. If, for instance, strychnin is to be examined for, the liquid is alkalinized, shaken out with chloroform, and the latter evaporated, when the alkaloid, in a condition of greater or less purity, is left as a residue; if morphin is sought, the alkalinized liquid is extracted with amyl-alcohol or acetic ether; and so on for the other individual poisons.

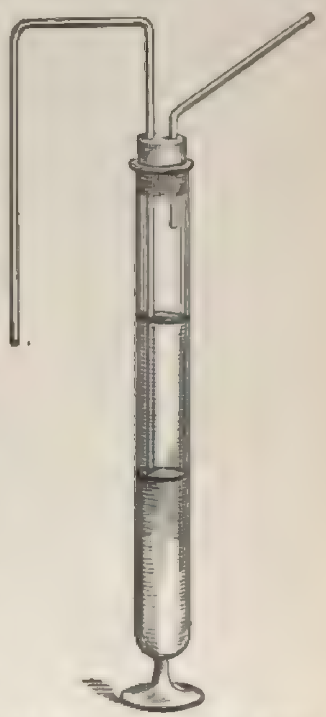


FIG. 28.—Prescott separating device.

The use of strong alcohol for the initial extraction, as is generally recommended, is inadvisable; it produces an abundant, firm coagulum of proteid material which is practically certain to occlude more or less of the alkaloids present and retain them in spite of repeated washings. This is especially true if an organ like the liver or kidney is operated on; but even in the case of softer material, like the contents of the stomach or of the intestine, particularly if a large amount of undigested food is present, a part of the alkaloid is almost sure to resist extraction. By using, however, as directed above, gradually increasing strengths of alcohol, the precipitation of the proteids is accomplished fractionally and

the danger of loss is reduced to a minimum. The step in which there is the greatest possibility of loss is the first extraction, and the use even of dilute alcohol is sometimes prejudicial. This is particularly the case when the amount of poison present is small and the material to be operated on bulky and of firm consistence. In this event the first extraction should be made with acidulated water, repeatedly applied, and the filtrate after evaporation to a small bulk treated with dilute alcohol, the remainder of the process being carried on as already directed.

The evaporation of considerable volumes of aqueous and alcoholic liquids at a moderate temperature, as contemplated in this and other methods, is often exceedingly tedious; it may be hastened, however,

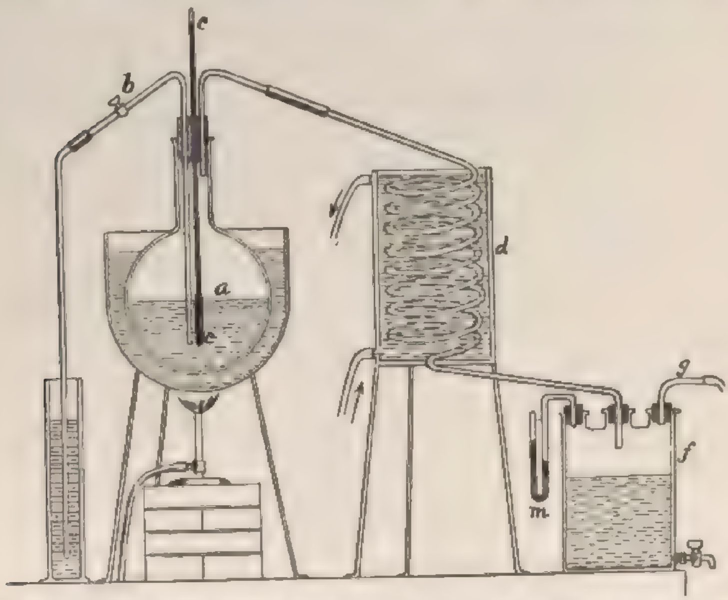


FIG. 29.—Apparatus for evaporating under reduced pressure (Ogier). The liquid is placed in the flask *a*, heated in a water bath, the temperature is indicated by the thermometer *c*. Tube *b*, provided with a stop-cock, is for admission of small bubbles of air to facilitate boiling; the tube also serves to introduce fresh quantities of liquid without opening the apparatus. *d* is a worm surrounded by running water. *f* is a bottle to receive the condensed alcohol; tube *g* communicates with a vacuum pump, the pressure is indicated by the manometer, *m*.

very advantageously by conducting the process under reduced pressure. An excellent apparatus for accomplishing this is described by Ogier,[1] and is shown slightly modified in Fig. 29.

Separation of Alkaloids from Residual Fat.—In all of the above processes for separating alkaloids, during the evaporation of alcoholic solutions and also when the residue from an alcoholic extraction is treated with acidulated water, there is nearly always a considerable separation of fatty material which has a tendency to retain more or less of the alkaloid, and unless means are resorted to for recovering the latter a considerable loss may occur, amounting in some cases to nearly,

[1] *Traité de chimie toxicologique*, p. 508.

if not quite, half of the total amount present. A number of methods have been suggested to avoid this loss. Repeated agitation with warm acidulated water may be resorted to; but perhaps the best means is that recommended by Noyes,[1] which consists in dissolving the oily residue in purified ligroin, adding very dilute sulphuric acid, and thoroughly mixing the two by passing them through a filter with the aid of a pump. The acid solution, which contains now practically all the alkaloid, and the ligroin are allowed to separate and the former added to the other portion of the aqueous extract.

Separation of Ptomains.—One of the most serious obstacles in the way of the perfect identification of alkaloids when extracted by the above processes is their possible contamination with ptomains and other products of putrefactive decomposition. While this fact applies to all of the alkaloids more or less, it is particularly true of morphin, whose reactions in certain respects are closely simulated by those of some of the proteid decomposition-products.[2] For strychnin, fortunately, we have a means in the use of warm concentrated sulphuric acid of getting rid of such extraneous substances; but this method is not practicable with morphin or with most of the other alkaloids. Many methods have been suggested for the elimination of these bodies; the following is a process which I have used with much advantage, and which generally gives satisfactory results. It depends on the fact that ptomains and other products of proteid decomposition when exposed to the air, especially if gently heated, slowly take up oxygen, resinify, and otherwise change so as to lose their identity; they become in part difficultly soluble in acidulated water, and in part removable from an acid fluid by repeated shaking with ether, while most alkaloids under this treatment undergo comparatively little or no decomposition.

The process is carried out as follows: The ether, chloroform, amyl-alcohol, or other immiscible solvent used in extracting the alkaloid is allowed to evaporate in a capacious watch-glass or flat-bottomed evaporating-dish so that the residue may present a large surface to the action of the air. After the solvent has evaporated, the residue is gently heated on a water-bath for an hour or two, and afterward put aside in a warm place for two or three days. The more or less resinified material is then extracted with very dilute sulphuric acid, the solution filtered, the acid filtrate shaken repeatedly with ether, alkalinized, and shaken out with the appropriate immiscible solvent. The latter is allowed to evaporate, and if the residue is still found to be impure, the treatment above described is repeated one or more times, sometimes many repetitions being necessary. Eventually, if care is taken with all the details of the process, the alkaloid is usually obtained in a practically pure condition. The method, however, entails the loss of more or less material, and this should be remembered in quantitative testing. But for qualitative work this is usually of little moment, as a small

[1] *Jour. Amer. Chem. Soc.*, 1894, vol. xvi., p. 109.

[2] See p. 691, in section on Ptomains and Other Bacterial Products in Their Relation to Toxicology.

amount of a pure alkaloid is greatly to be preferred to a large quantity of impure—the former yielding delicate and characteristic reactions, while the latter often gives entirely unreliable results.

Another method of extracting alkaloids which is of particular importance in this connection is the—

Process of Kippenberger.[1]—To isolate alkaloids and glucosids, and especially to separate them from ptomains and other products of putrefaction, this process is possessed of much merit.[2] The method is based on the fact that the alkaloids and glucosids are generally soluble in a mixture of tannic acid and glycerin, while proteids and putrefactive substances in general are usually rendered insoluble by it. The process is as follows:

The finely subdivided material is macerated for two days at 40° C. in a 10 per cent. solution of tannic acid in glycerin. The mass is then placed in a cloth bag and the fluid portion thoroughly squeezed out, preferably by means of a press; the liquid thus obtained is heated to 50° C., cooled, and filtered. The filtrate is shaken with petroleum ether to remove fats, and is then heated on a water-bath to expel a small amount of petroleum ether left in solution. The liquid is now agitated thoroughly with successive portions of chloroform, first while still acid, and then after being alkalinized with potassium hydroxid. Sodium or potassium bicarbonate is next added to convert any excess of alkaline hydroxid into carbonate, and the mixture extracted with chloroform containing about 10 per cent. of alcohol. The solution is then saturated with sodium chlorid and shaken out with chloroform containing about 15 per cent. of ether.

The *petroleum ether* in the above process extracts traces of jervin and veratroidin, together with fat.

The *chloroform* extracts from the *acid solution* aconitin, cantharidin, colchicin, digitalin, geissospermin, jervin, narcotin, papaverin, picrotoxin, and traces of brucin, delphinin, narcein, strychnin, and veratrin.

The *chloroform* removes from the *alkaline solution* apomorphin, atropin, brucin, codein, coniïn, emetin, nicotin, pilocarpin, spartein, strychnin, veratrin, and any narcotin and papaverin left from the preceding operation.

The *alcohol-chloroform* extracts morphin and narcein, and the *ether-chloroform* removes strophanthin.

Upon evaporating either of the above immiscible solvents the substances it has extracted from the liquid are left as a residue to be examined by appropriate tests.

In order to avoid some of the inconveniences attending the use of glycerin in the above process, Kippenberger[3] has more recently suggested the use of acetone in conjunction with tannic acid. To the solution thus obtained a small quantity of glycerin and a little dilute hydrochlo-

[1] *Zeitschr. f. anal. Chemie*, 1895, vol. xxxiv., p. 294.

[2] Consult also p. 707, in section on Ptomains and other Bacterial Products in Their Relation to Toxicology.

[3] *Zeitschr. f. anal. Chemie*, 1900, vol. xxxix., p. 627.

ric acid are added, the acetone evaporated off, and the resulting aqueous solution is extracted by immiscible solvents, as described above.

Separation of Poisons by Dialysis.—Taking advantage of the discovery by Graham of the difference in diffusibility through porous membranes of crystalline and colloid bodies, many poisons of a crystalline nature may be separated from contaminating organic matter by the process of dialysis. When first suggested for this purpose it was believed that it would yield results of great value, but practical experience has scarcely borne out these expectations. At times it may be used with considerable profit in selected cases or in preliminary testing, but, as a rule, it cannot compare in value with the methods already given for the separation of either mineral or organic poisons. Since in using the process, however, nothing is done to the material to change chemically its constituents, the poison is procured in practically the same condition in which it existed in the matter submitted to examination. In some instances this is a point of no small moment in toxicologic investigation; it is occasionally of great importance, for the purpose of identification, to establish the exact form in which a poison has been administered, or in which it exists in the stomach-contents or vomited matter, and when such is the case, dialysis may be of much service.

The usual method of performing the operation is to place the material, finely subdivided and brought to a thin consistence by the addition of water if necessary, in a dialyzing vessel, which is supported in a dish of pure water for twenty-four hours with an even level of the two liquids. At the end of that time the crystalline constituents of the material, such as arsenic, tartar emetic, oxalic acid, and the various alkaloids, are generally found, in part at least, in the outer fluid, and may be tested for directly by the use of appropriate reagents or may be recovered for identification by evaporation. In the case of alkaloids the liquid may be shaken out with immiscible solvents for the purpose of more complete purification. If the amount of poison in the material, however, is small, or if it has entered into firm proteid combination, so little of it may pass into the outer fluid as to give unsatisfactory results.

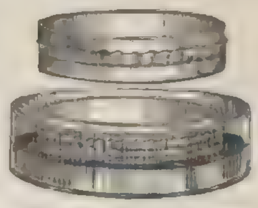


FIG. 30.—Graham's apparatus for the application of dialysis.

The dialyzer above spoken of may be made by tying a piece of thoroughly sound parchment-paper over a hoop of wood or gutta-percha, or over the end of a glass jar or stout beaker from which the bottom has been removed. It may be made of any size suitable for the amount of material used, but it is well to have as large a surface of parchment-paper as possible to facilitate the operation. Usually a diameter of 6 to 8 inches (15 to 20 centimeters) and a height of 2 to 3 inches (5 to 8 centimeters) will serve the purpose (see Fig. 30).

INORGANIC POISONS.

While it is convenient to classify together the poisons derived from the mineral kingdom because they have a common origin in inorganic nature, a better reason is found in their similarities in chemical behavior and in the formation of their compounds. The mutual neutralization of acids and alkalis furnishes additional ground for this association. Another basis for so grouping them is found in the fact that almost all are irritants. Among them will be considered oxalic acid, which is not of mineral origin, but has a local irritant action and strongly acid chemical properties. The mineral poisons may be subdivided into *corrosives* and *irritants*, both of which produce a local organic mischief. Sometimes all are considered as irritants, and two classes are recognized: first, those which coagulate albuminous substances and thereby cause an eschar; and second, those which soften and dissolve the tissues, causing swelling and discoloration. In this classification the first group would include the corrosive acids, with oxalic and carbolic acids and such metallic irritants as corrosive mercuric chlorid. The primary local effect upon mucous membrane is a dry, cloudy, grayish-white condition, though the secondary effect of the acids is to cause yellow or brown discoloration. On the other hand, the members of the second group, composed of the corrosive alkalis, produce swelling and clearing up of the mucous membranes, with a dark color due to the imbibition of altered hematin by the submucous structures. When poisoning is due to carbolic acid or mercuric chlorid, the color of the blood in the affected parts shows through as a bright brick red.

CORROSIVE POISONS.

The local action of this class of poisons is destructive. Applied to the skin, they have disorganizing effects analogous to those of a burn, and which, if extensive, may produce fatal consequences. Brought in contact with the mucous membranes, they have here a like destructive action; if swallowed, any part of the alimentary tract may be corroded, and the lining membrane of the larynx become the seat of acute inflammation with dangerous effusions.

The altered tissues are easily perforated and the contents of the stomach or intestines may escape into the peritoneal cavity. Death may ensue within a few hours from shock or the suffocation due to dropsical closure of the opening of the glottis.

Short of perforation, the corrosion may cause such wide-spread breaking down of the gastric glands as to annul their digesting power.

An additional cause for slow starvation is found when the esophagus is narrowed, if not closed, by inflammatory exudations.

The **symptoms** come on at once and are well defined. In the mouth there is burning pain, with a strong taste characteristic of the class to which the poison belongs: sour if an acid, soapy if an alkali, and metallic if due to a metallic salt.

Immediately intense pain is felt in the throat and stomach, soon involving the whole abdomen. Vomiting and purging supervene, the discharges often showing blood, pure or altered by chemical action. There is thirst, with painful swallowing. The voice is faint and husky from the inflammation of the epiglottis and the larynx.

Stated broadly, the **postmortem appearances** are those of local softening or of hardening and contraction, with disintegration of tissue, either in circumscribed areas or largely distributed and surrounded by acutely inflamed tissues. A superficial erosion of the mucous membrane may be seen in one place, while in another the basement membrane may be involved, and in another a patch of the entire structure give way, leaving openings into other parts. The differences in chemical action cause peculiar colorations, such as the yellow stain made by nitric acid (see Plate 2) and the brownish-black imbibition of altered hematin caused by sulphuric, hydrochloric, and oxalic acids, and also by the alkaline hydroxids (see Plate 7). The corrosive poisons are subdivided into the *corrosive acids* and the *corrosive alkalis*.

CORROSIVE ACIDS.

The mineral acids, sulphuric, nitric, and hydrochloric, are sour liquids which turn red the vegetable blue colors, such as litmus, and change the hue of dyed clothing mostly to red or yellow, while they injure the texture. When concentrated, they rapidly destroy organic substances, and on the living body cause the most violent pain. They render the alkalis neutral and dissolve the common metals with effervescence. They are simple corrosives causing well-marked symptoms, due solely to their action on the part to which they are applied. In the United States the mineral acids are often taken by suicides, but as criminal poisons they do not figure in our records. In England and on the Continent they are frequently resorted to by the homicide.

The organic corrosive acid, oxalic, is a sour solid which may be given as an intensely acid solution, lacking the fierce dissolving power of the strong mineral acids and causing symptoms some of which are local and common to other corrosives, but others also which are remote and specific in character, due to cerebrospinal involvement.

The antidotes owe their power to chemical neutralization, changing the fiery acids to harmless neutral salts.

Calcined magnesia, given freely suspended in water or milk, is a perfect antidote. When it cannot be had at once, as promptness is all-important, prepared chalk, whiting used to polish silver, plaster scraped from the wall, soapsuds, or largely diluted alkalis, such as sodium car-

bonate ("washing-soda"), sodium bicarbonate ("bread or baking soda," "saleratus"), sodium hydroxid ("concentrated lye"), or the corresponding compounds of potassium, should be given in milk or water. The soapsuds and alkalis are of no value against oxalic acid, as they form soluble alkaline oxalates which are absorbed and produce systemic poisoning.

It rarely happens that the antidote is given soon enough to prevent the energetic action of the poison, and even after thorough neutralization it would be best to give milk and very dilute alkaline solutions for some hours. As the tube of the stomach-pump or the siphon impinging upon the softened structures may do irreparable harm, it must not be used,[1] though later on the esophageal stricture may call for careful treatment by dilator and tubes.

SULPHURIC ACID.

(Chemical formula, H_2SO_4; Synonym, *Oil of Vitriol.*)

There are few processes in the arts and manufactures that do not use at some stage the "oil of vitriol." It can be had at any chemists in common trade, and is used for cleansing metals as a household article. In countries where the law makes it difficult to purchase the arsenical or alkaloidal poisons, the ease with which sulphuric acid can be procured makes it a very common poison in use by the poorer classes for suicidal purposes, in spite of the pain that characterizes its action. It is rarely given in food for homicidal purposes, because it betrays the poisoner by the altered appearance of the charred food, by the stains on the clothing, lips, and tongue, by the fiery taste, and by the characteristic symptoms. It has been given to young children and even to adults in the form of medicine, taken, as disagreeable doses usually are, from a spoon back of the tongue, so as to avoid tasting.

Poisoning has occurred from the accidental substitution of it for oils, syrup, or glycerin. It has been poured into the ear, given by enema, and even injected into the vagina. From this poison there were 49 deaths in England and Wales for the five years from 1883 to 1887 inclusive. From journals ordinarily accessible there have been collected[2] 303 cases, of which 207 were fatal, 84 recovered, 45 were homicidal, 151 suicidal, and 75 accidental.

Local External Effects.—Malicious persons resort to it to disfigure the face or ruin the clothes of a rival by throwing a quantity of it at the hated person. Occasionally in chemical laboratories, while experimenting with it, flasks containing it will burst and the contents be dashed into the face of the experimenter. If it strikes the eye, blindness may result. In contact with the skin it causes great agony and a lasting scar. Instant action is necessary to prevent these serious effects. Water must be applied freely, the whole face immersed in a basin of it or held under a running tap, and the eyes opened under the

[1] Compare p. 310, under General Principles of Toxicology.

[2] Witthaus and Becker, *Medical Jurisprudence, Forensic Medicine, and Toxicology,* New York, 1896.

water. A paste of sodium bicarbonate or a piece of soap will help to neutralize the residual acid at the burned points. The burn may be treated afterward with *linimentum calcis*. It is a common accident in the laboratory for the acid to fall upon the clothing. If not promptly touched with ammonia or some alkaline solution, the spot turns red and soon becomes rotten.

Properties.—Pure, strong sulphuric acid of a specific gravity of 1.84 contains 96 per cent. of real acid (H_2SO_4), and is a colorless, heavy, oily liquid, not fuming, odorless, extremely sour, combining actively with water, and blackening or charring organic substances. The commercial "oil of vitriol" is not so pure, being colored light-brown by suspended carbonaceous matter and containing small amounts of dissolved metals, principally lead and arsenic. When added to water, heat is given out. If the proportion of the mixture is 3 of acid to 1 of water, the temperature will rise above 212° F. (100° C.). It has the property of abstracting water from the air, 100 grains under favorable conditions absorbing 120 grains of water in four days. This great affinity for water explains the charring action upon organic matter from which it abstracts the elements of water while dissolving all but the black carbon. When the concentrated acid is heated with zinc, copper, or other metals, the gas sulphur dioxid is liberated; if the acid be dilute, then if any action occurs, the gas evolved is hydrogen.

"Nordhausen acid" is a form manufactured in Bohemia and used largely in chemical manufactures. It is a dark-brown, heavy, oily, fuming liquid, with a specific gravity of 1.9. Its formula is $H_2S_2O_7$, and it is regarded by some as a solution of SO_3 in H_2SO_4. Two weaker forms are used in medicine, the *dilute*, of 10 per cent., H_2SO_4, and the *aromatic*, of 20 per cent., H_2SO_4. It is applied externally as a powerful caustic in the shape of Ricord's paste, made with powdered charcoal, and Michel's paste, made with powdered asbestos.

Symptoms.—On the instant of contact with the mouth there is intense local pain, extending down the throat and gullet to the pit of the stomach, along the track of the acid. The tongue swells until it fills the mouth, and is covered with a white coating. Later it may be a corroded and shapeless mass.

The saliva flows profusely, but cannot be swallowed without pain, if at all, owing to the pharyngeal inflammation. Gasping and a hoarse voice denote that some of the acid has touched the larynx and caused spasmodic closure of the glottis.

The thirst is extreme, and is accompanied by persistent retching and vomiting. The ejected matter is very sour and slimy, often bloody and loaded with portions of the mucous membrane of the gullet and stomach. The face has an agonized and anxious expression, the eyes look hollow, the nose is pinched and cold, the skin is clammy, the pulse is feeble, the breathing is difficult, and the extremities are convulsed. The case may end fatally in a few hours or after several days by asphyxia, stupor, or convulsions. When perforation of the stomach is caused by rapid solution of its walls, the symptoms of fatal collapse

rapidly develop and death is comparatively painless. When death is not so sudden and the inflammatory symptoms subside, the unfortunate one has a lingering death of starvation from stricture of the gullet or of the pylorus, and an incurable dyspepsia due to destruction of the coats of the stomach. Mendelssohn[1] reports the case of a woman of twenty-five years of age, who took, for suicidal purposes, a moderate amount of sulphuric acid, and, surviving the immediate effects, had symptoms of gastric ulcer and enteric fever. After four weeks improvement began, but was arrested in the sixth week by attacks of vomiting. To relieve the pyloric stenosis resection was resorted to, but death followed in twelve hours. The gastric mucous membrane was almost wholly destroyed, the pylorus narrowed to the caliber of a small probe.

Fatal Dose.—The smallest fatal dose reported as given to an adult[2] is 60 grains (3.8 gm.). Death ensued in a child of one year after 20 drops.[3] It is difficult to state the minimum limit of fatality, owing to the fact that much depends on the part touched by the acid and much on the amount of food present in the stomach. Even the smallest amount would be permanently injurious if it reached the gullet, causing narrowing of the food-channel. Few, if any, infants survive this poison, and of the adult cases, the mortality is two-thirds.[4]

Fatal Period.—In the infant quick inspiratory effort sometimes carries the poison into the larynx, and immediate death may ensue from spasmodic closure of the glottis.[5] The shortest period recorded for the adult is one hour. Most cases die within twenty-four or thirty-six hours; some die from sequels after weeks, months, or years. In 124 fatal cases, the reports of which were studied by Witthaus,[6] 13 died in less than three hours; 42 in three to twenty-four hours; 28 in one to seven days; 17 in one to four weeks; 16 in one to three months; and 8 in more than three months.

Treatment.—Three objects are to be kept in view: First, prompt neutralization of the acid; second, weakening by dilution; third, relief of the asphyxia, which sometimes threatens life immediately. For neutralization, magnesia and chalk are the best, but in an emergency soap-suds, whiting, or wall-plaster (an impure calcium carbonate) will serve the purpose. Weak alkaline solutions of sodium or potassium carbonate may be used with caution, as great distress, if not injury, to the weakened walls is possible from the stomach distention due to the liberation of large quantities of carbon dioxid gas. All the antidotes must be given suspended or dissolved in large quantities of water or milk. In the absence of a neutralizing antidote water alone must be used immediately and in large drafts, followed by raw eggs. Should symptoms of asphyxia appear as a result of laryngeal implication, then tracheotomy or intubation must be resorted to at once. Morphin may be

[1] *Charité Annalen*, 1887. [2] Christison, R., *A Treatise on Poisons*, Edinburgh, 1836.
[3] Taylor, A. S., *On Poisons*, 1875.
[4] Guy and Ferrier, *Principles of Forensic Medicine*.
[5] Thomson, *Lancet*, 1836–37.
[6] Witthaus and Becker, *Medical Jurisprudence, Forensic Medicine, and Toxicology*, New York, 1896.

given hypodermically to relieve pain, and nutritive enemata to support life. The sequels—perforation, collapse, contraction of the gullet, gastritis, and impaired digestion—must be treated by appropriate measures as the occasion arises.

Postmortem Appearances.—The *primary* pathologic changes found when death occurs within a few days are those of acute disorganization of the structures of the mouth, gullet, stomach, and neighboring parts. The lips and tongue are softened and eroded; the throat and gullet, whitish or gray in color, the first effect of the acid on mucous surfaces being to coat them with a white paint of altered secretion and membrane; the stomach is brown-red, due to imbibition of altered hematin or black from charring, its mucous lining loose in shreds or patches, the folds large and deep from swelling, sometimes softened so as to tear under gentle manipulation; the peritoneum may be blackened from perforation; the duodenum, red and thickened.

The *secondary* pathologic changes, seen when death follows after several weeks of chronic illness from some of the sequels, are ulceration of the gullet and contraction of its caliber from scars; the stomach is stripped of mucous membrane, partly or wholly red, its capacity much reduced by contraction, its walls thickened and adherent to neighboring parts.

Tests.—*Barium Chlorid Test.*—It is customary to test for sulphuric acid and the soluble sulphates by first acidulating with hydrochloric acid to prevent a precipitate being produced by the salts of certain other acids, such as carbonic, phosphoric, and oxalic, and then adding a solution of barium chlorid which throws down the white barium sulphate. The free acid, in common with other acids, reddens litmus, turns cochineal yellow, and decolorizes red phenolphtalein.

As proof of the presence of a *free mineral acid* litmus will not serve, as it is affected by acid salts and by the organic acids of digestion. Resort can be had to paper colored by certain anilin dyes which react to minute quantities of free mineral acids, but not in the same way to the organic acids nor to acid salts. A drop of the gastric contents containing a free mineral acid put on Congo-red paper leaves a dark-blue spot, while organic acids in large amount give a violet color; on tropaolin paper freshly prepared from an alcoholic solution (1 : 1000) it leaves a red-brown spot which changes to lilac when gently heated; on paper dipped in fresh solution of 1 gm. of phloroglucin and 2 gm. of vanillin in 30 gm. of alcohol it turns red when heated in a capsule. A weak solution of methyl-violet, distinctly violet in hue, is turned blue by adding a very weak mineral acid.

Charring Test.—When sulphuric acid is applied undiluted to white paper it darkens, and if gently heated chars, the paper; even if largely diluted, by heating the paper so as not to scorch it, the water evaporates and the acid will reach the charring-point. In some degree this property is shared by hydrochloric acid.

Veratrin Test.—A drop of the free acid will turn the alkaloid veratrin yellow, and finally an unchanging crimson. When the free acid is

a valued escharotic, but is not suited for internal administration, the dilute form, containing 10 per cent. of HNO_3, being more eligible.

Properties.—The concentrated pure acid of the United States Pharmacopeia is a colorless, fuming, heavy liquid, with a specific gravity of 1.41, containing 68 per cent. of HNO_3. On prolonged exposure to light and air the peroxid and other lower oxids of nitrogen are developed and impart a yellow color. It is then called nitroso-nitric or fuming acid. The *aqua fortis* of commerce is weaker, having a specific gravity of 1.36, is usually colored yellow, and fumes with the orange-colored vapor of mixed lower oxids. Most of the metals dissolve in it, but gold and platinum are exceptions. It corrodes organic matter by oxidation, not by carbonizing, as sulphuric acid does. Animal matter is turned a deep yellow, the color of picric acid. Albumin is coagulated by it, and if the acid is strong, the white coagulum turns the characteristic yellow. It gives promptly the acid reaction with litmus and other color indicators.

Symptoms.—The records of the comparatively few cases show no important difference from the symptoms produced by sulphuric acid and already described, with the exception of the color of the mouth and lips, which, with nitric acid, is intensely yellow, though at first the parts are blanched and white. There are intense pain, vomiting, thirst, and great depression. Eructations of gas are frequent and distressing, due to its direct development by the action of the acid on organic substances.

Fatal Dose.—Three drams by the mouth in an adult have destroyed life,[1] but a much smaller quantity would suffice to cause fatal suffocation from spasmodic closure if it were to enter the larynx, as it is likely to do in children.

Fatal Period.—The average duration of life is about twenty-four hours; the shortest time reported in the case of an adult[2] was an hour and three-quarters, while a case is recorded[3] in an infant who died in a few minutes. In some cases death has been delayed for weeks, months, or years, the remote effects of the poison then proving fatal. In 27 fatal cases reported Witthaus[4] states that 2 died in less than three hours, 10 in three to twenty-four hours, 6 in one to seven days, 3 in one to four weeks, 3 in one to three months, and 3 in more than three months.

Treatment.—The extraordinary energy and rapidity of action of nitric acid make it difficult to administer antidotes with sufficient promptness to be of much help. It is always advisable to use chalk, whiting, magnesia in milk, soapsuds, and eggs as antidotes, with the hope of neutralizing some free acid. The method is the same as for sulphuric acid and for the corrosive acids generally. In all there is instant local death of parts struck by the poison, rapidly followed by inflammation of surrounding viscera. Our antidotes cannot restore the

[1] Warren, *Records Boston Soc. for Med. Imp.*, 1853.

[2] A. S. Taylor, *On Poisons*, 1875. [3] *Ibid.*

[4] Witthaus and Becker, *Medical Jurisprudence, Forensic Medicine, and Toxicology*. New York, 1896.

POISONING WITH CONCENTRATED NITRIC ACID (VON HOFMANN).

A suicide aged thirty-six years swallowed a large quantity of nitric acid at 9 A. M., and died at 1 P. M. the same day. The body revealed yellow discoloration of the skin about the mouth and of the epithelial lining of the mouth. The epithelium of the esophagus was bright yellow and arranged in thickened, rigid folds. The epithelium was easily detached, showing a pale yellowish membrane with black vessels. The stomach was contracted, and its mucous membrane was changed to a bright yellow, fissured eschar. The submucosa, exposed in places, was ragged, partly yellow and partly black through the imbibition of blood. The duodenum and other organs adjoining looked as if cooked and were yellowish in color.

PLATE 2.

tissues to health, nor can they diffuse into distant parts fast enough to be of much avail. The symptoms must be treated on general principles as they appear.

Postmortem Appearances.—All the parts to which the acid is applied present the various marks of erosion—in places hardening and thickening, in others ulceration and sloughing, general pulpiness, shreddy mucous surfaces denuded of membrane, and perforations of the gullet, the stomach, or the intestine. The most characteristic pathologic change is the permanent citron-yellow or orange-brown color of the tissues acted on (see Plate 2).

Tests.—Even when very largely diluted—*i. e.*, to 0.2 per cent.—the acid reddens litmus. (See tests for free mineral acids, p. 345.)

Copper Test.—Poured upon slips of copper and gently heated, effervescence occurs and red-brown vapors arise that redden moist litmus-paper. If the amount of nitric acid is small, the color of the fumes may not be noticed, and a more delicate test is required. By holding in the vapors a piece of paper moistened with potassium iodid and starch paste, a blue color develops.

Brucin Test.—Upon a crystal of brucin a drop of nitric acid strikes a blood-red color; upon morphin an orange hue, with orange-colored fumes.

Ferrous Sulphate Test.—Upon a white porcelain surface put a few drops of the suspected liquid, a drop of sulphuric acid, and a crystal of ferrous sulphate; the crystal turns dark green, and finally brown. Even the combined acid in nitrates yields the same proof with any of the above tests, provided pure sulphuric acid is first used to free the nitric acid. If eggs have been given as an antidote, the nitric acid must be taken from the albumin by means of a solution of potassium carbonate; the resulting soluble nitrate can then be treated by equal parts of sulphuric acid and water before applying the above tests.

Detection.—On inspection the stains left on the clothing will be found dry and partaking of the same citron-yellow change found on the skin or other animal tissue touched by this acid. The yellow stain produced by tincture of iodin will be discharged by potassium hydroxid or by ammonia water, but the nitric-acid stain is indelible; ammonia and the alkalis only intensify it to an orange hue. If the piece of stained cloth is boiled in some distilled water, litmus-paper will reveal the acid reaction. When the acid liquid is neutralized with potassium carbonate, filtered, and evaporated to dryness, crystals of potassium nitrate form. When these crystals are dissolved in water and a drop of pure sulphuric acid is added, the nitric acid is set free and strikes a blood-red color with brucin, or yields ruddy fumes with copper turnings, or responds to any other test for nitric acid.

If the vomited matters are decidedly acid, the acidity should be measured by titration with decinormal solution of sodium hydroxid. The resulting sodium nitrate can then be tested by treating with sulphuric acid and applying any of the tests above mentioned.

As nitrates are not constituents of ordinary food or of the animal

tissues, it is proof enough if these are found in any amount above a trace. It is not necessary to make a quantitative analysis. The vomited matters or the tissues should be extracted with boiling distilled water and potassium carbonate, and then filtered. Crystals of potassium nitrate are obtained on evaporation which respond to all the tests given above for nitrates.

Fumes of Nitric Acid.[1]—The emanations of nitric acid are a mixture of nitric acid vapor with various lower oxids, all of them offensive and irritating to the air-passages. In the manufactures mentioned above as making use of this acid these vapors may do great harm if the processes are not carried on in closed vessels and the noxious fumes passed into milk of lime. The habitual breathing of air containing only a small amount frequently leads to severe chronic bronchitis with general impairment of health. In the annals of toxicology cases of acute poisoning are reported from chemists suddenly inhaling the fumes rising when a carboy of the acid has been accidentally broken. The symptoms are like those of capillary bronchitis. In the cellar of Jefferson Medical College is a supply-room in which carboys are kept. By accident a carboy of nitric acid had been left unstoppered, and after the summer vacation, the room having been closed for some weeks, a whitewasher worked in it for several hours until he was forced by illness to give up. He suffered from tightness of the chest, difficult breathing, dry cough, headache, nausea, and physical prostration, the symptoms not subsiding for a week or more.

In fatal cases there is found usually congestion of the larynx, trachea, and bronchial tubes, and sometimes edema of the lungs or effusion of blood. Although the effects appear to be mainly those of direct irritation, some cases show inflammatory changes in the lining of the right auricle. Acute cases should be treated by fresh air and inhalations of ether to relieve the sense of constriction.

HYDROCHLORIC ACID.

(Chemical formula, HCl; Synonyms, *Muriatic Acid; Spirit of Salt.*)

In the ordinary acceptation by physicians, druggists, and manufacturers, this name is given to a liquid which, properly speaking, is a strong aqueous solution of the true acid, itself a gas imparting to the water its own chemical properties. By accident or for suicidal purposes it caused 90 deaths in the five years in England and Wales reported by the Registrar General. In New York city between 1870 and 1891 inclusive there were 6 cases of accidental death from this source. Out of 68 cases reported in medical journals, Witthaus[2] found 55 were fatal, 5 homicidal, 39 suicidal, and 24 accidental.

Properties.—Commercial hydrochloric or muriatic acid is a transparent, yellow, corrosive liquid. Its strength or percentage of pure acid gas is approximately the product of 200 and the decimals of the

[1] Consult also p. 676, section on Gaseous Poisons.

[2] Witthaus and Becker, *Medical Jurisprudence, Forensic Medicine, and Toxicology*, New York, 1896.

specific gravity. Thus a sample of a specific gravity of 1.15 should contain 30 per cent. HCl (200 × 0.15).

The chemically pure acid is colorless, the yellow color of the commercial article being due to a trace of iron from the apparatus used in its manufacture. A more important contaminant is arsenic, taken from the sulphuric acid used in generating it. The average amount of this impurity is 0.25 per cent. of arsenic trioxid. The pure acid liquid of the United States Pharmacopeia is sour, of pungent odor, and contains 450 volumes of gas dissolved in 1 volume of water, which increases more than one-third in bulk. It contains 31.9 per cent. by weight of the gas. The *dilute* acid of the pharmacopeia contains 10 per cent. by weight of the anhydrous acid. On exposure to the air the strong acid gives off visible fumes due to the union and condensation of the invisible gas with the aqueous vapor of the air. The fumes have a pungent odor, an acid taste, are irrespirable, are one-fourth heavier than the air, and when allowed to blend with the fumes of ammonia, form dense white clouds of ammonium chlorid. The acid dissolves most of the metals, but not gold and platinum, and when heated with manganese dioxid, chlorin is set free. It is the natural acid of the gastric juice, and is used with pepsin as an aid to digestion. It is employed in chemical analysis as a group-reagent, from its having the property of precipitating mercury (from mercurous salts), lead, and silver.

Symptoms.—This acid is very corrosive, like sulphuric and nitric acids already considered, but not so severe in its local action as either of the others. Owing to its volatility there is great liability of acute laryngeal inflammation from its irritating fumes, although the liquid itself may not enter the glottis. The lips, tongue, and throat are first white, but later become brown and rotten. There are instant pain in mouth, throat, and abdomen, difficult swallowing, husky voice, spasmodic breathing, retching and vomiting, feeble pulse, and general weakness, the mind remaining clear to the last. If the patient survives these acute symptoms, he remains subject to stricture of the gullet or pylorus, with loss of function of the stomach.

Fatal Dose.—As with the other acids, a few drops may prove fatal if they enter the larynx. By rapid swallowing and quick transmission to the stomach death may follow upon a fluidram dose.[1]

Fatal Period.—From the acute effects death may ensue in fifteen hours or even in two hours, but, as a rule, the duration of life will be twenty-four hours. The secondary consequences are productive of a poor vitality for a variable period. One case has been reported of death from stricture of the pylorus after four months. Witthaus[2] found that in 40 fatal cases reported in medical literature 2 died in less than three hours, 18 in three to twenty-four hours, 6 in one to seven days, 4 in one to four weeks, 6 in one to three months, and 4 in more than three months.

[1] *Brit. Med. Jour.*, 1871.

[2] Witthaus and Becker, *Medical Jurisprudence, Forensic Medicine, and Toxicology*, New York, 1896.

Treatment.—The remedial measures are the same as for sulphuric and nitric acids, the acid being neutralized by magnesia, chalk, plaster, soapsuds, or alkaline bicarbonates, given with milk, water, and raw eggs. The effects must be combated by general therapeutic rules.

Postmortem Appearances.—The pathologic changes found after death cannot be distinguished from those induced by sulphuric acid except by the local effects on lips and face.

Hydrochloric acid leaves no permanent stain nor erosion externally, while sulphuric acid discolors and nitric acid turns yellow. Internally we find the signs of intense inflammation, with a shriveled and worm-eaten condition of the mucous membrane, which has a white or brownish color. The appearances due to sulphuric acid are the same except that the destruction of tissue is greater, but the yellow marks of nitric acid are always characteristic.

Tests.—The free acid gives the acid reaction to litmus. (See tests for free mineral acids, p. 345.) A glass stopper or rod wet with it and held near an open bottle of ammonia water smokes with the white clouds of ammonium chlorid. Poured upon zinc it evolves hydrogen gas; if heated with manganese dioxid it yields greenish-yellow chlorin gas, which bleaches a piece of moist litmus-paper suspended in the vapor.

Silver Nitrate Test.—The chief test for chlorids serves equally for this acid—*i. e.*, silver nitrate, which gives a heavy, curdy, white precipitate of silver chlorid, soluble in ammonium hydroxid, but insoluble in nitric acid.

Detection.—Very little help is derived from a study of the stains on clothing. At first a reddish spot appears. On some black dyes the color is greenish, but owing to the volatility of the acid, the spots are evanescent. They are not moist, charred, nor rotten as they are from sulphuric acid, nor are they yellow as from nitric acid. After a few days the moistened cloth will not affect litmus, but if boiled in water, silver nitrate will show more chlorids in it than in the untouched cloth.

In the examination of the vomited matters we are liable to a fallacy from the natural presence of 0.2 per cent. of hydrochloric acid in the gastric juice, and from the chlorin in the alkaline chlorids of food.

If the material is strongly acid, sulphuric acid must first be tested for and excluded. Distillation will then collect the volatile hydrochloric acid, which can be estimated by titration with sodium hydroxid.

To determine both free acid and the combined chlorids, first make a filtered watery extract and divide it into two equal parts. One of these is neutralized by adding excess of sodium carbonate, which fixes the volatile free acid. Both are evaporated to dryness, the unneutralized portion losing all its free acid. Both residues are redissolved in water and are treated separately with acid solution of silver nitrate. If the neutralized portion shows more chlorids than the other, the difference equals the amount of free hydrochloric acid originally present in each portion. In this analysis 100 parts of silver chlorid precipitated represent about 80 parts of hydrochloric acid (specific gravity 1.15) or 25.43 parts of the anhydrous acid.

Nitromuriatic Acid.—By mixing 1 part of nitric and 3 parts of hydrochloric acid the commercial *aqua regia* is prepared. This is an unstable liquid, evolving free chlorin and other gases, and eventually becoming much weaker than when first made. It dissolves all the metals, including gold and platinum, and oxidizes iodin, phosphorus, and sulphur. It coagulates albumin, turns it yellow, and finally dissolves it, as it does all vegetable and animal substances, with the production of ruddy fumes.

While the dilute acid is given internally as a medicine, the concentrated acid is an exceedingly corrosive poison, the symptoms and postmortem appearances of which differ from those of nitric acid in degree only. The antidotes are the same as for the other mineral acids.

OXALIC ACID.

(Chemical formula, $C_2H_2O_4 2H_2O$; Synonym, *Acid of Sugar.*)

Oxalic acid and its salts are widely present in nature, being found in various plants, such as rhubarb (used for pies), nightshade, dock, sorrel (used for greens), and in animals also, occurring not infrequently as a constituent of the human urine. In the latter it is incidental to the gouty condition and some forms of dyspepsia, occurring as calcium oxalate in the form of a whitish deposit made up of microscopic crystals, octahedral or dumb-bell shaped, and insoluble in warm water and in acetic acid.

It can be prepared from sugar by oxidation with nitric acid, and, therefore, is sometimes known in the arts as "acid of sugar." Its bleaching properties and solvent powers for metallic oxids make it useful to dyers and workers in leather, makers of straw hats and bonnets, and workers in marble and in brass. About the home it is used to remove ink-stains from linen. Druggists dispense it at low price, and consequently the would-be suicide not infrequently resorts to it. Its resemblance to Epsom salts leads to accidental poisoning, but the very sour taste is likely to betray the homicide, who rarely resorts to it except when it can be masked by some other sour beverage. In the five years from 1883 to 1887 inclusive there were registered in England and Wales 120 cases.[1] There were 14 accidental deaths from it in New York city in the twenty-one years from 1870 to 1891 inclusive.[2]

Of 169 cases found by Witthaus[3] reported in medical literature previous to 1895, 83 were suicides, of whom 41 were accidental. In the decade, 1871–80, in England and Wales, out of 1000 suicides by poison, 159 took oxalic acid.

Properties.—The crystals of oxalic acid are colorless, four-sided, prismatic, not deliquescent, and so closely resemble in appearance those of magnesium sulphate and zinc sulphate that it is often confounded with them. These crystals are very acid, soluble in about 10 parts of

[1] *Registrar-General's Reports.* [2] *Report of the New York City Board of Health.*
[3] Witthaus and Becker, *Medical Jurisprudence, Forensic Medicine, and Toxicology,* New York, 1896.

cold water and in 2½ of cold alcohol, but very sparingly in ether. Heated on porcelain or platinum, it sublimes without residue.

It can be distinguished from the substances for which it is sometimes mistaken by the following ready tests, applicable in the household :

	Oxalic acid.	Magnesium sulphate.	Zinc sulphate
Taste	Sour.	Bitter, nauseous.	Bitter, metallic.
Reaction	Very acid.	Neutral.	Slightly acid
Heated	Sublimes.	Fixed.	Fixed.
Sodium carbonate	No precipitate, but effervescence.	No effervescence, but a white precipitate.	No effervescence, but a white precipitate.
Ink	Bleaches.	No effect.	No effect.

Potassium Binoxalate (Chemical formula, KHC_2O_4,H_2O).—This salt is usually dispensed by druggists to remove rust and ink-stains from linen, to bleach straw, and to polish metals, under the very deceptive name of "essential salts of lemon" and "salts of sorrel," and sometimes without even the "grim heraldry of death" usually blazoned on labels for poisonous substances. It is sometimes dispensed as a white powder, although it crystallizes in colorless rhombic prisms; it has a decidedly acid reaction and sour taste, and is soluble in 40 parts of water. It is likely to be mistaken for cream of tartar, which is also a sour white solid. Almost equal to oxalic acid in the violence of its poisonous action, its symptoms, postmortem appearance, antidotes, and detection are practically the same.

Symptoms.—What is said of the toxic effects of oxalic acid is applicable also to potassium binoxalate. While the symptoms vary considerably in different cases, they can be conveniently classified as, first, those due to the *local* erosive action on the mucous surfaces, and, second, those arising from the *remote* impression upon the nervous system—convulsive and narcotic. The symptoms produced by the local action of a strong solution and a large amount are very sour taste, thirst, pain and burning in mouth, throat, and stomach, difficult swallowing, vomiting of black or bloody substances, collapse. Occasionally pain is absent. Sometimes death may occur without vomiting.

"If," says Christison,[1] "a person immediately after swallowing a solution of a crystalline salt which tasted purely and strongly acid is attacked with burning in the throat, then with burning in the stomach, vomiting, particularly of bloody matter, imperceptible pulse and excessive languor, and dies in half an hour, or still more in twenty, fifteen, or ten minutes, I do not know any fallacy which can interfere with the conclusion that oxalic acid was the cause of death. No parallel disease begins so abruptly and terminates so soon; and no other crystalline poison has the same effect."

Hart[2] reports a case of oxalic-acid poisoning in a boy aged fifteen years. Twelve minutes after the poison had been swallowed the patient was unconscious, his skin pallid and clammy, and his extremities cold. The radial pulse could not be felt. The pupils were fairly dilated. The jaw was fixed in tetanic spasm, and froth exuded

[1] Christison, *Treatise on Poisons*, 1836. [2] *Lancet*, October 1, 1898.

from between the teeth. One-tenth of a grain of apomorphin was injected hypodermically; a stomach siphon-tube was introduced after the jaws had been forced apart, and a pint of warm water was placed in the stomach, but immediately expelled. Vomiting continued, and consciousness returned. The boy was given a half-ounce of powdered chalk, suspended in water, and this also was shortly ejected. Recovery proceeded under stimulation. The quantity of poison taken was upward of 2½ drams.

If, owing to the smallness of the dose, death is not prompt, absorption of the poison ensues, and then the remote or *neurotic* symptoms appear. These are headache, cramps, convulsions, delirium, and coma. If the patient survives, there may be numbness and tingling, with loss of voice, lasting for weeks. When a small dose has been taken in dilute solution, the symptoms have not come on for hours, and then the nervous phenomena are more prominent.

Fatal Dose.—The least weight of the acid recorded as having fatal consequences is 1 dram (3.88 grams).[1] Statistics show that the dose most likely to prove fatal is from half an ounce to an ounce. Early vomiting and a measure of relief are caused by excessive doses. More than an ounce (14.2 grams), if retained, usually causes death, although recovery has occurred after a dose of 2 ounces.[2]

If efficient antidotes are instantly given, there may be recovery from much larger doses, although the majority of cases prove fatal.

Fatal Period.—In one case death, supposed to be due to gastric hemorrhage, occurred without pain in *three minutes*.[3] In other cases surviving the acute action on the alimentary tract death has occurred from coma after several days, one living until the twenty-third day. Witthaus[4] states that of 74 fatal cases, 62 lasted less than twenty-four hours; in 12 the victim was found dead, in 5 death occurred in less than ten minutes, in 15 in from ten to thirty minutes, in 9 in from one-half to one hour, in 2 in two hours, in 10 in from two to thirteen hours, in 4 in one and two days, in 4 in from two to five days, in 5 in from five to fourteen days.

Treatment.—The chemical antidotes are finely divided chalk or calcined magnesia or its carbonate, suspended in a large quantity of water, and followed by free drafts of warm water to facilitate vomiting. As the toxic action is prompt, the antidote must be given at once. With a shovel or a kitchen knife the wall-plaster can be scraped off and used as an impure calcium carbonate.

Oxalic acid is chemically neutralized by the alkalis as well as by the alkaline earths (lime and magnesia), but the alkaline oxalates being soluble and poisonous are inadmissible, while the oxalates of calcium and magnesium are insoluble and innocuous. Emetics may be necessary, but the stomach-pump is likely to injure the eroded lining of the gullet and stomach.

[1] *Lancet*, December 1, 1885. [2] Tapson, *London Med. Gazette*, vol. xxxi., p. 142.
[3] Ogilvie, *Lancet*, 1845, vol. ii., p. 205.
[4] Witthaus and Becker, *Medical Jurisprudence, Forensic Medicine, and Toxicology*, New York, 1896.

Postmortem Appearances.—Colored stains upon the lips and face are absent, but the lips, tongue, throat, and gullet are usually white, and the lining membrane is loose, eroded in patches, and contracted into folds. Sometimes the stomach is black from extensive venous engorgement and contains blood or a brownish, grumous material; sometimes the membrane is pale and smooth, or detached in shreds; sometimes red, with the black veins strongly marked and corrugated. While deep erosions are not uncommon, it is rare to have complete solution of the walls of the stomach so as to cause the symptoms of perforation during life. Both peritonitis and pleuritis have been found as complications, and perforations of the stomach also, but these last in some cases have been supposed to be due to changes after death. The kidneys are congested and loaded with oxalates.

Tests.—A solution of oxalic acid or of potassium binoxalate reddens litmus-paper.

Calcium Test.—Either of them yields, with excess of calcium hydroxid, acetate, or sulphate, a white precipitate of calcium oxalate, insoluble in ammonia or acetic acid, but soluble in strong hydrochloric or nitric acid.

Silver Nitrate Test.—Either of them gives with silver nitrate a copious white precipitate of silver oxalate, soluble in ammonia and in nitric acid, while silver chlorid would be insoluble in the nitric acid. The silver oxalate, dried and heated on platinum, disperses with a slight explosion and a white smoke.

Lead Acetate Test.—With lead acetate a white precipitate of lead oxalate is formed which is soluble in nitric acid, but insoluble in acetic acid.

Potassium Permanganate Test.—Mixed with potassium permanganate and dilute sulphuric acid the oxalic acid is decomposed, and the permanganate, slowly losing its color, is converted into manganese sulphate.

Sublimation Test.—Heated on platinum foil, the acid crystals slowly sublime at as low a temperature as 100° C. (212° F.), and they are entirely and promptly dissipated at 150° C. (302° F.). The potassium binoxalate does not sublime, but changes to potassium carbonate, which effervesces when touched with an acid, and turns red litmus-paper blue.

Detection.—The symptoms of corrosive poisoning from an acid liquid which has left no colored spots upon the skin would be significant. A strong solution makes on black cloth a dark-brown, uncorroded spot, which gives the oxalic acid reactions. The vomited matters should be searched for the leaves of sorrel or green material of the rhubarb pie; not that these are ever fatal, but so as to exclude the possibility of a complication in the analysis. In the vomited matters and gastric contents the acid will be partly free, partly combined as soluble oxalate, and partly combined as the insoluble calcium or magnesium oxalates. If it should be mostly free, the following method will serve:

1. Having made an extract with hot dilute hydrochloric acid and filtered it, add lead acetate, which will throw down the lead oxalate along

with various other lead compounds. This deposit should be suspended in water and hydrogen sulphid passed through it for two hours. The oxalic acid is set free in solution, the black lead sulphid being thrown down. After separation by a filter the filtrate should be tested with calcium acetate.

2. If the oxalic acid is in the combined state, the following is the better method: Digest the suspected matters with warm dilute hydrochloric acid until the mixture is quite fluid, filter, and to the filtrate add ammonium hydroxid until an alkaline reaction is reached. After standing the liquid is decanted and the deposit collected on a filter. This deposit is calcium oxalate. The filtrate mixed with the decanted fluid is treated with excess of calcium acetate and the precipitate separated on a filter. This second deposit represents the free acid in the original material. To determine the nature and amount of the first deposit, it should be washed with acetic acid on the filter and afterward put into a beaker and dissolved by cautiously adding strong hydrochloric acid and gently heating. Excess of ammonia will precipitate it completely if sufficient time is allowed. After decanting the clear fluid the deposit is washed by decantation, put into a tared dish, dried in a water-bath, and weighed. If this deposit is calcium oxalate, it will be white, and when a portion is heated on platinum, leave a gray ash of calcium carbonate. Another portion warmed in a test-tube with strong sulphuric acid evolves carbon dioxid gas, which can be identified by conducting it through a delivery tube into baryta water. A third portion, suspended in water slightly acidulated with sulphuric acid, will discharge the purple color of potassium permanganate. This test can be applied by standard volumetric solutions and an estimate of quantity obtained.

If the poison has been taken as neutral sodium or potassium oxalate, the local symptoms and pathologic changes may not be at all characteristic. The effects come on after absorption and are mainly systemic. To make a complete examination, the poison must be looked for outside the alimentary canal, by separating it from the urine and the finely divided tissue of the kidney. The method would be the same as that for vomited matters containing the combined acid.

CORROSIVE ALKALIS.

Under this heading will be considered the hydroxids or hydrates of potassium, sodium, and ammonium. It is well to note that their basic *carbonates* also are not only strongly alkaline in reaction, but in concentrated solution have a corrosive effect. The action of the corrosive alkalis is chemical or local, and limited to the part with which they come in contact. This corrosive power is due to their solvent action on albumin, their saponifying property when mixed with fatty matter, and their avidity for the water of the tissues. They cause rapid and deep destruction of the animal structures. The local symptoms are like those of the corrosive acids. The general symptoms are likewise those of the shock of a violent lesion added to the immediate consequences of the

lesion due to its locality. According to Falck,[1] out of 27 cases, 22 died (81.5 per cent.). Poisoning from them is most often accidental, though, according to the reports of Viennese hospitals, they are not infrequently taken for suicide.[2]

Reports of 81 cases of poisoning by the corrosive alkalis and carbonates have been studied by Witthaus,[3] who found 41 due to accidental mistake and 17 to suicide. He found 9 cases of poisoning from sodium hydroxid, 3 from sodium carbonate, 1 from sodium bicarbonate, 21 from potassium hydroxid, 12 from potassium carbonate, 2 from a mixture of potassium hydroxid and carbonate, and 33 from "concentrated lye."

Upon the salts of the alkaline metals neither hydrogen sulphid nor ammonium sulphid has any visible effect. Unlike the alkaline earths, they are not precipitated by ammonium carbonate.

POTASSIUM HYDROXID.

(Chemical formula, KOH; Synonyms, *Potassium Hydrate; Caustic Potash.*)

Properties.—The pure substance is a gray-white solid with an angular fracture. It imparts a soapy feeling when handled, has a soapy taste, and a strongly alkaline reaction to litmus. Heated, it melts to a colorless liquid; run into cylindric molds it makes *potassa fusa*, the ordinary form seen in the shops. It dissolves in half its weight of water, evolving heat; it is soluble also in alcohol and glycerin, but insoluble in ether. It deliquesces rapidly, and in the moist state freely takes up carbon dioxid gas to make potassium carbonate.

Pharmaceutic Preparations.—*Potassa*, or caustic potash, occurs in cylindric rods. *Liquor potassæ*—solution of potash—is a colorless, acrid, alkaline, corrosive liquid with a specific gravity of 1.036, and containing about 5 per cent. of potassium hydroxid. *Potassa cum calce* (Vienna paste) is made of equal parts of potassa and quicklime. *Potassa carbonas impura* (pearlash), under the name of potashes, used for cleansing oil-lamps, occurs as a dark mass, deliquescent, strongly alkaline, and caustic. *Potassa carbonas pura* occurs as white crystals, deliquescent, alkaline, and caustic.

Symptoms.—Taken in strong solution, a large dose of caustic potash or the carbonate will cause a nauseous, soapy taste, accompanied by burning pain in mouth, throat, and stomach. Vomiting of alkaline bloody material soon follows, and later colicky pains and great abdominal tenderness with purging of shreds of epithelium, mucus, and blood. The lips and tongue swell and turn brown, swallowing is difficult, and the voice is hoarse. The pulse becomes feeble and rapid, the skin cold and damp, the breathing hurried and shallow. Surviving these symptoms, the patient may die after some days of starvation from stricture of the gullet.

[1] Falck, *Lehrb. d. prak. Toxikologi*, 1880.
[2] Hofmann, *Lehrb. d. gericht. Med.*, fifth edition, 1891.
[3] Witthaus and Becker, *Medical Jurisprudence, Forensic Medicine, and Toxicology*, New York, 1896.

Fatal Dose.—The ordinary fatal quantity is half an ounce, but 30 grains have proved sufficient.

Fatal Period.—From the acute effects death may come in three hours; from the secondary effects, the final event may be delayed for weeks or even years. The average duration is about twenty-four hours.

Treatment.—The chemical antidotes are weak acids and oils. The most convenient weak acid is vinegar, but diluted lemon-juice or orange-juice will serve. Milk, olive-oil, melted butter, or lard would also neutralize the alkali, though not so promptly. The stomach-pump is not admissible.[1] The pain will call for morphin injections; collapse should be met by stimulants, and threatened starvation by nutritive enemata.

Postmortem Appearances.—The mouth, throat, and gullet are whitish and softened. The stomach and intestines are bright red or black from extravasated blood; the lining membrane disorganized and stripped in patches. The secondary pathologic changes seen when death closes the history of a chronic case are denudation of the lining membrane, ulceration, and points of stricture in gullet or pylorus.

Tests.—With excess of tartaric acid the potassium salts in strong solution give a colorless precipitate of potassium bitartrate, and with platinum chlorid a yellow precipitate insoluble in alcohol. To make these tests conclusive any ammonium salt must be removed by boiling with calcium hydroxid. To a colorless flame they impart a violet hue, and viewed by the spectroscope there is seen a characteristic combination of a dull-red band with a faint violet line.

Detection.—As alkalinity of the gastric contents has never been reported in any normal case, the mere fact that vomited matters or gastric contents have an alkaline reaction would be so exceptional as to be suspicious. After separating the soluble alkali from the undissolved matter by dialysis, the clear liquid should be titrated with decinormal sulphuric acid and tested for potassium. As the chlorid, sulphate, and phosphate of the metal are natural constituents of the food and of the body itself, more or less of these will be found always present. Hence if the fluid is not alkaline, the process must include quantitative determinations of the different metals. If the analyst can obtain a sample of the substance taken or a piece of the clothing stained, his task is much simpler.

SODIUM HYDROXID.

(Chemical formula, NaOH; Synonyms, *Sodium Hydrate; Caustic Soda.*)

Under the name of "concentrated lye," an impure mixture of the hydroxid and the carbonate is largely used as a rapid cleanser in the laundry and in the making of soap. The author saw a case of a child of four years who, in playing about the laundry, out of curiosity ate some of the contents of a can containing "lye." The poison apparently did not reach the stomach, but corroded the throat and left a stricture

[1] Compare p. 310, under General Principles of Toxicology.

STOMACH AFTER POISONING BY SODIUM HYDROXID (VON HOFMANN).

A suicide aged twenty-one years took a large quantity of sodium hydroxid (40 per cent. solution). He had "swollen mouth" and gastro-enteritis, with vomit of blackish masses. Vinegar was given as an antidote; emesis was favored and the stomach washed out, yet he died in two days. The mucous membrane of mouth and esophagus was softened, congested, and swelled, with detachment of the epithelium in patches. The plate shows the stomach, which had lost its epithelium, leaving coarse folds of a brownish-black, soft, and swollen mucous membrane. The brown color was due to the imbibition of alkaline hematin. The patches of black dots indicate points of hemorrhagic infiltration.

PLATE 3.

of the gullet, which permitted swallowing of liquid food only. The patient slowly wasted away from starvation. There was no autopsy.

Properties.—The *hydroxid* occurs in gray-white masses or in molded sticks closely resembling potassium hydroxid. Like that it is strongly alkaline in reaction, soapy in taste, fuses by heat, dissolves freely in water with evolution of heat, is deliquescent, and in the moist state absorbs carbon dioxid from the air, forming the carbonate. This carbonate is not deliquescent like potassium carbonate, but efflorescent. When a can of caustic soda is opened, the solid will first liquefy, then absorb carbon dioxid, and finally solidify in a whitish powder.

Sodium carbonate, under the name of "sal soda" or "washing soda," is used as a domestic article to soften water and assist in cleansing. It occurs in rhombic octahedrons or in large angular masses which effloresce and crumble to powder. It is alkaline, acrid in taste, soluble, and caustic in every respect like the corresponding salt of potassium, only less severe.

Symptoms.—The symptoms are those of a corrosive poison, differing in degree only from those caused by potassium hydroxid and carbonate.

Fatal Dose.—About the same as for the potassium compounds.

Fatal Period.—The duration of life will depend on the dose and the lesions, and may be described as about the same as that given for the potassium compounds.

Treatment.—The antidotes are vinegar and lemon-juice to neutralize the alkali, and milk, oil, or butter to saponify it.

Postmortem Appearances.—The toxic effect is purely local. Although less active than the potassium compounds, the caustic forms of soda dissolve the albumin of the tissue, abstract the moisture, saponify the fatty material, and corrode deeply and widely (see Plate 3).

Tests.—Unlike the potassium salts, the salts of sodium are not precipitated by tartaric acid nor by platinum chlorid. Its common salts are all soluble. With a fresh solution of *potassium antimoniate* they yield a white precipitate. To a colorless flame they impart an intense yellow color, which the spectroscope places as the bright D-line.

Detection.—The history of the case, inspection of inflamed spots on the face and hands, the strong soapy taste, and marked alkaline reaction of vomited matters will go far to prove a caustic alkali. The above given tests can be applied to determine the character, making allowance for the sodium chlorid always present in food and tissue. As commercial sodium hydroxid nearly always contains a small quantity of arsenic, a trace of the latter would strengthen the evidence in favor of the caustic alkali.

AMMONIUM HYDROXID.

(Chemical formula, NH_4OH; Synonyms, *Ammonium Hydrate; Ammonia Water.*)

Under the names of "hartshorn" and "ammonia," ammonium hydroxid is largely used in the household as a cleansing agent to remove paint, oil, and dirt generally from clothing. The following case which came under the observation of the author will show how easy it is to have acci-

dental poisoning from it. An adult, unable to sleep, took from a closet a bottle containing a colorless fluid, which he supposed to be a solution of potassium bromid; the room being dimly lighted, he did not read the label, but took a tablespoonful of what proved to be ammonia-water. He detected the poison as soon as it was tasted or smelt and before it was swallowed. For about a week his mouth and tongue were raw, swollen, and painful; he was unable to masticate or swallow solid food.

The liquefied ammonia gas (NH_3) is used in ice-factories and refrigerators. Sometimes the receivers burst and the vapors fill the room, with deadly consequences to those who are exposed to them.[1] Sometimes, to arouse fainting persons, it is given too strong by inhalation. In the five years from 1883 to 1887 inclusive, 45 fatal cases were registered in England and Wales. Of 50 cases caused by aqua ammoniæ, Witthaus[2] found that 29 were accidental, 18 suicidal, and 3 homicidal. Of 47 cases, 27 died and 20 recovered.

Properties.—Ammonia gas (NH_3) is a colorless gas having a pungent odor, irritating to the eyes and the mucous lining of the air-passages, turning moist red litmus-paper blue. Under a pressure of 6½ atmospheres it is condensed to the liquid used in ice-machines to create a freezing temperature by its evaporation. *Ammonium hydroxid* is a strong solution of the gas in water. Water will absorb 700 times its volume at ordinary temperatures and thereby acquire the alkalinity, the pungency, and the chemical properties of the gas itself.

Pharmaceutic Preparations.—*Aqua ammoniæ fortior* contains 28 per cent. by weight of ammonia gas, has a specific gravity of 0.900, and is a powerful corrosive. *Aqua ammoniæ* has 1½ times more water, only 10 per cent. of ammonia gas, and a specific gravity of 0.960. *Spiritus ammoniæ* is a solution of the gas in alcohol, of the same strength as the aqua, and better adapted for internal use. When it has added to it the carbonate, with small quantities of oils of nutmeg, lemon, and lavender, the *aromatic spirits* is produced. *Ammonii carbonas* occurs as whitish angular masses, giving off the characteristic irritating and alkaline vapor of ammonia, and caustic in strong solution.

Symptoms.—The nature and gravity of the effects will depend greatly on the strength of the solution, and on whether or not the subject received a strong dose of the vapor by the lungs. The direct chemical action upon vital tissue is the same as that of potassium hydroxid, though less in degree—*i. e.*, the albumin is dissolved, the fatty matter saponified, and the water abstracted. The respiratory symptoms are a suffocative feeling due to spasm of the glottis, followed by a sense of pain and weight in the chest, with an irritative cough due to inflammation of the larynx and trachea.

The symptom due to the local caustic effect of the fluid is burning pain in mouth and throat, extending to the stomach if the poison went so far. There are salivation, vomiting, and difficulty in swallowing. As

Medical and Surgical Journal, 1887.

Medical Jurisprudence, Forensic Medicine, and Toxicology,

a result of a free absorption of the poison by the lungs and stomach cases display grave remote effects sometimes with great rapidity. The heart's action is sometimes arrested in a few minutes; sometimes there is immediate unconsciousness with coma, and death in a few minutes; sometimes there is unconscious delirium, soon ending in death.

A typical case is that reported by J. M. DaCosta:[1] A man aged forty-six years, took by mistake a large quantity of strong aqua ammoniæ, but swallowed only a small portion. In a short while the breathing was frequent and stertorous, the voice husky, the glottis, uvula, tonsils, lips, gums, and tongue all swollen. There were headache and delirium, enlarged cervical lymphatics, cough with bloody expectoration. The urinary symptoms were remarkable, the secretion being diminished, but dense, alkaline, highly albuminous, showing separate blood-cells and many tube-casts, epithelial, hyaline, and granular. The serious symptoms soon subsided and convalescence set in after four days.

Fatal Dose.—A teaspoonful of the stronger aqua ammoniæ has in at least one instance proved fatal,[2] and two fluidrams have caused death in two or three other cases. Recovery, however, has sometimes followed much larger doses, such as a tablespoonful, and even upward of a fluidounce has been taken without fatal results.

Fatal Period.—By suffocation and syncope death has occurred in four minutes after inhalation of the gas. On the other hand, death may occur after many months as the result of the starvation due to stricture of the gullet or pylorus.

Treatment.—The antidotes are very weak vinegar, lemon-juice, oil, butter, and milk. The sequels are to be treated as they arise.

Postmortem Appearances.—These are not markedly different from the inflamed state of the alimentary tract as caused by the other caustic alkalis. When the vapor acts as an irritant upon the air-passages, an inflamed state of the larynx and even of the bronchi may be found.

Tests.—Ammonia gas turns red litmus-paper blue, and makes a white smoke when mixed with the fumes of a rod wet with hydrochloric acid. All the salts are volatile when heated, and evolve the gas spontaneously or when heated with calcium hydroxid. Platinum chlorid yields a yellow precipitate like that given by potassium.

Detection.—Owing to the volatility of ammonia, its hydroxid and its carbonate, these soon escape from the body. During life, or soon after death, detection is easy by the characteristic odor. If the volatile preparation has been fixed by the antidote, the vapor can be developed by heating the material with lime. This vapor will be alkaline and form white fumes with hydrochloric acid.

If the organic material to be examined is putrid, allowance must be made for the ammonia produced by putrefaction. This is never enough to develop the dense white fumes of ammonium chlorid from a rod wet with hydrochloric acid. The amount may be estimated by distillation, neutralizing the distillate with hydrochloric acid, evaporating nearly to dryness, and precipitating the double chlorid of ammonium

[1] *Boston Medical and Surgical Journal*, 1891. [2] *Guy's Hospital Reports*, 1871.

and platinum by adding excess of alcoholic solution of platinum chlorid. After filtration, the precipitate is washed with alcohol, dried, and weighed; 100 parts represent 8.6 parts of ammonia (NH_3).

IRRITANT POISONS.

In the class of irritants are placed a large number of poisons that figure prominently in the bills of mortality. Some of them are of animal and some of vegetable origin, but the present study is limited to those derived from the mineral kingdom. They cause pain in the throat, gullet, and stomach, great thirst, nausea and vomiting, abdominal tenderness, purging and straining, with bloody stools, scanty, and often albuminous, urine. After death the alimentary tract shows an inflamed condition not very characteristic except when it involves the entire length from mouth to rectum, an extent never found in the specific inflammatory affections of the stomach and bowels, such as cholera morbus and acute gastric or intestinal catarrh. There are redness and swelling of the mucous glands, dark patches of infiltrated blood, softening, and, as a secondary result of the inflammation, ulceration and perforation (see Plate 5). With those minerals that add remote effects to the local ones just given there are symptoms and pathologic changes referable to the specific organs affected, as the heart, liver, and kidney.

PHOSPHORUS.

(Chemical symbol, P.)

This element is a constituent of the tissues and fluids of the human body, occurring largely in the bones as calcium phosphate and in the nervous centers as a compound with fat and albumin. Ever since it was first used to tip lucifer matches its poisonous properties have been known; indeed, on the Continent of Europe it has been the favorite rat poison. While the other active poisons are guarded by law from general distribution, this one is easily obtained as the heads of matches and as "rat-paste," which contains from 1 to 4 per cent. of phosphorus mixed with oil, flour, sugar, and coloring-matter.[1] It is rarely used by homicides, but frequently by suicides, and sometimes children ignorantly eat the paste or suck the heads of matches. In the five years from 1883 to 1887 inclusive there were registered in England 71 deaths from poisoning by phosphorus and matches.[2] More than half the cases were in children. Of the adults, nearly all were suicidal, a few only being accidental and none criminal. In spite of the garlicky taste and smell, it could be given in coffee, the more easily if at the same meal onions or garlic had been eaten. Out of 522 cases studied by Witthaus,[3] in only 10 was the

[1] *Coster's Rat and Roach Exterminator* contains 2.13 per cent. of phosphorus, and though the buyer is assured by the label that it is "not poisonous," two fatal cases have been reported from taking it. *Parson and Co.'s Vermin Exterminator* has 0.4 per cent. of free phosphorus.

[2] *Repts. of Registrar-general.*

[3] Witthaus and Becker, *Medical Jurisprudence, Forsenic Medicine, and Toxicology,* New York, 1896.

substance anything but match-heads or vermin-poison: 110 were homicidal, 294 suicidal, 66 accidental, 4 taken to cause abortion, 1 by experiment, 2 by quacks. Of the 110 homicidal cases, only 3 were in the United States; of the 294 suicidal, 179 were in Germany and 3 in the United States; of the suicidal cases, 172 were women, many of whom probably took it to produce abortion.

Properties.—The *ordinary crystalline* or *waxy* form usually occurs in translucent cylinders which cut like wax, and when kept under water turn yellow and become coated with a thin white crust. As it oxidizes in the air it should be kept under water that has been well boiled. It takes fire at 122° F. (50° C.), a temperature easily reached by friction between the fingers, hence the caution to handle it with forceps. If it should take fire in the hand, it will burn severely, and at the same time more or less of the poison will be absorbed. The poison in the burn should be made inert by a lotion of chlorinated soda or a paste of chlorinated lime.

It has the odor and taste of garlic, is very sparingly soluble in water, slightly soluble in alcohol and glycerin, but freely so in carbon bisulphid, almond oil, and ether. Under water at 111° F. (44.5° C.) it melts to an oily fluid, which can be run into cylindric molds. Exposed to the air, white fumes of its lower oxid are evolved, and in the dark emit a feeble light.

Red phosphorus is an allotropic form, made by heating the ordinary form in a closed vessel without air for thirty-six hours. It is a red-brown powder which does not oxidize in air, need not be kept under water, and requires a much higher temperature to inflame it than does the waxy form. The pure red phosphorus is not poisonous, but the commercial article sometimes contains as much as 0.6 per cent. of the waxy, poisonous form.

The *lucifer matches* commonly sold are tipped with waxy or poisonous phosphorus mixed with potassium chlorate, sand, and glue, but the "*safety*" *match* is tipped with potassium chlorate and antimony sulphid without phosphorus. In order to light the "*safety*" match it must be rubbed upon the side of the containing box, which is covered with a thin coat of red or non-poisonous phosphorus, mixed with sizing.

Pharmaceutic Preparations.—Phosphorus is usually given in a pill-mass with cocoa-butter or some other excipient and coated with sugar or gelatin to prevent oxidation. The presence of the free phosphorus can be shown by cutting the pill open and exposing the mass to gentle heat in the dark. It should "phosphoresce"—*i. e.*, emit light. *Oleum phosphoratum* is a solution in almond oil of 1 per cent. strength. *Spiritus phosphori* is a solution in absolute alcohol of about 0.1 per cent. strength. An *ethereal* solution is also used.

Phosphine (Chemical formula, PH_3).—*Phosphorus terhydrid* or *phosphureted hydrogen* when inhaled is a very poisonous gas, reducing the oxyhemoglobin of the blood. It can be made by boiling phosphorus with strong potash or soda lye, or by generating hydrogen in the presence of phosphorus or its lower oxids. It is colorless, sparingly soluble,

and, as ordinarily made, inflames spontaneously on contact with the air. When evolved with hydrogen it burns with a greenish flame, but if dry and insufficiently supplied with air, the flame is white. When passed through a solution of silver nitrate, the silver is deposited as metal, leaving nitric and phosphoric acids in solution; by adding excess of molybdic acid the phosphoric acid can be detected.

Symptoms.—If the phosphorus be taken in lumps, the effect is not proportionate to the weight. To be fully effectual it must be dissolved or finely divided, as it is in the rat-pastes and pill-masses.

The cases of poisoning are often referred by their symptoms to one of the three classes established by the researches of Tardieu[1]—a common form, showing symptoms of local irritation and jaundice; a hemorrhagic form like scurvy, in which jaundice and effusions of blood occur; and a nervous form, in which jaundice is accompanied by creeping sensations, cramps, drowsiness, delirium, and convulsions.

Nearly 90 per cent. of the cases suffer from *acute irritation* followed by *jaundice and profound blood changes.* Complaint is made that the substance taken had the taste and odor of garlic. Sometimes violent pain in the throat, gullet, and stomach is experienced immediately, accompanied by vomiting and purging. The breath is phosphorescent, and the ejected matters may be bloody, garlicky in odor, and emit light when stirred in a shallow dish. In a large number of cases there is an interval of several hours between the taking of the poison and any symptom whatever.

Death from collapse may come at this early stage, but usually the irritation abates and *jaundice* sets in after a period of comparative comfort. This quiet interval usually lasts from two to three days, but it may be only one day in length or be prolonged for several weeks. The jaundice portends more or less profound *blood changes.* In addition to the general effects wrought by the biliary matters in the circulation, there will be the toxic symptoms, caused by the presence of phosphorus derivatives. Given in detail, there will be yellowness of skin and conjunctiva and tenderness over the liver, with an increased area of hepatic dulness. Headache, insomnia, and itching eruptions of the skin are common. The urine is saffron-yellow or olive-green in color from the presence of bile-pigments, scanty, albuminous, bloody, containing tube-casts and occasionally leucin and tyrosin. Extreme weakness culminating in *heart failure* is a characteristic due to the degenerations of the muscular tissue, including the heart. These stormy signs soon culminate in delirium, convulsions, coma, syncope, and death.

In a certain proportion of cases, not necessarily fatal, the toxic effects on the blood and its vessels are made conspicuous by the *hemorrhages* which accompany the jaundice. Blood may be effused under the skin in spots or pass out by one or more of the mucous channels. Hemorrhage has occurred from the nose, mouth, bowels, kidneys, and bladder all at once. Women will have uterine hemorrhage, and if pregnant, will abort, with alarming flooding. Anemia and exhaustion reach an

[1] Tardieu, *Étude Médico-légale*, etc., 1867.

extreme stage, and delirium ending in death may supervene after months have elapsed since the administration of the poison. Even when the direct influence of the poison has passed away and life is no longer threatened, there may be persistent debility and local palsies.

The rarest form of acute poisoning is that in which the *nervous phenomena* are the most conspicuous. This form is likely to occur when the case is one of inhalation of fatal quantities of phosphorus vapor. In the preparation of "rat-paste" or in the making of matches the materials may be accidentally heated so as suddenly to evolve large quantities of phosphorus vapor. The effects are fainting attacks, succeeded by profound prostration with extreme muscular weakness.

Emphasis must be laid upon the variety of the symptoms, permitting of many different clinical pictures, and also upon their insidious development. There can be but little doubt that at one time many cases were incorrectly diagnosed as acute yellow atrophy of the liver. This is not surprising, as the history of the case after the liver symptoms appear is the same as in acute yellow atrophy, even to the contraction of the organ itself. In a very small proportion of cases surviving a week jaundice does not occur. Casper reports a case that lived for twelve hours, the only marked symptoms being one act of vomiting and a garlicky odor of the breath, which was luminous in the dark.

West [1] reported a case of unusual delay in the supervention of fatal jaundice. A woman of fifty-two took phosphorus rat-poison. Within fifteen minutes she had burning in mouth and abdominal pain. Emetics and turpentine were given. In twelve days she was considered well. Four weeks later jaundice set in, with great depression, black vomit, and pains in the head, back, and legs. In six days she died. The autopsy revealed a large, firm, pale yellow, and fatty liver.

Fatal Dose.—In the treatment of nervous diseases the usual dose is one-fiftieth of a grain thrice daily, but some persons can bear gradual increase to as much as one-quarter of a grain. It would be risky to begin with these maximum quantities, as the subjects of nervous diseases are usually very susceptible. A lunatic under the observation of Lobel died from the effects of 0.116 gram (less than one-eighth grain).

A healthy adult would have his life put in jeopardy from one grain taken in a finely divided form, such as the pill, paste, or the match-head. A child is reported to have died from sucking the heads of two matches, containing about one-fiftieth grain of phosphorus. On the other hand, there has been recovery after ten packages of matches have been sucked.[2]

Fatal Period.—Death has occurred in less than one hour, but the duration of life is very diverse in different cases. Some die in four hours; three-fourths of the cases die within a week; some cases become chronic, the patient dying a lingering death after many months.

Treatment.—In considering the best remedial procedures it must be noted that great differences have been observed in the time of onset of the symptoms. In the majority they commence after an interval of

[1] *Lancet*, 1893. [2] Singer, *Prager med. Wochenschr.*, 1883.

from two to six hours; in a few they are described as immediate; in four-fifths they come on within six hours. In every case presenting a history of a poisonous dose the treatment should be instituted at once, instead of waiting for the symptoms to appear. There is need for instant evacuation by the stomach-tube and washing out of the stomach with a solution of *potassium permanganate* of the strength of 0.5 to 1 per cent. (about 4 grains to the fluidounce), leaving about a pint in the stomach. This antidote, introduced by Antal, of Budapest, has a chemical reaction with the phosphorus, by which the latter is said to be changed to harmless compounds. Potassium permanganate oxidizes the phosphorus, forming phosphoric acid and phosphates, itself changing to manganese dioxid. In the absence of a stomach-tube the antidote should be given—4 grains in an ounce of water, frequently repeated. The permanganate is in part reduced by the organic substances of the food, and hence the necessity of giving it in excess, although in a dilute solution to avoid gastric irritation. Quite recently Fr. Lanz has analyzed the results of treating 39 cases with potassium permanganate. He considers that these cases do not speak in favor of potassium permanganate as an antidote. Thornton has recommended a diluted hydrogen peroxid (1 to 3 per cent.) used for the same purpose as the potassium permanganate.[1]

Copper sulphate is often recommended as an antidote. When its solution is mixed with phosphorus in a test-tube, the phosphorus is seen to change instantly to black copper phosphid, which is not injurious. There is one drawback to its use. In the quantities recommended and needed for full antidotal effect (3 grains frequently repeated) the copper salt is a decided irritant and is likely to aggravate the gastro-enteritis or set up a violent one of its own.

Another antidote honored by text-book commendation is turpentine. It is said to combine with the phosphorus to produce phosphoroterebinthinic acid, a non-poisonous solid. To be efficient the article must be an old acid sample, and some enjoin that the French article alone is of any value. As old French turpentine is not the kind kept officially by druggists, it is practically out of the question.

After potassium permanganate or hydrogen peroxid has been freely used for the phosphorus in the stomach, evacuation of the bowels should be secured by the use of some old turpentine that has been kept for a long time on the shelf in doses of half a dram given in an emulsion with mucilage every half hour. As the phosphorus tends to adhere to the mucous folds of the small intestine, it is advisable to maintain purgation by giving the turpentine for several days.

Postmortem Appearances.—The general toxic effect of phosphorus is to induce a wide-spread degeneration of glandular and muscular tissue. This degeneration consists in the formation of fat in place of the true cellular tissue. It is presumable that those cases of death in which no change has been found postmortem would have yielded a different report if the microscope had been used to aid the naked eye. The *stomach*

[1] *Therap. Gaz.*, 1893, vol. ix., p. 8.

may be free from signs of disease, although, as a rule, there will be a fatty degeneration of the epithelial cells, with thickening of the mucous membrane, due to enlargement of the glands and an occlusion by large granular cells. This condition obtains in the intestines and is often associated with hemorrhagic foci and minute inflamed areas. These appearances are found also in diseases due to septic conditions of the blood.

Even at an early period the *liver* is the seat of fatty degeneration. If seen early, it may be enlarged, yellow, deficient in blood, and present a mottled section. Under the microscope the hepatic cells are found to lack definition and to be granular or filled with large fat-globules. When death follows a chronic history, the liver may be found atrophied and the changes more profound.

The capsule of the *kidney* is easily stripped. Under it are found hemorrhagic patches. The organ itself is enlarged, and its epithelial cells and vascular walls are infiltrated with granular fat.

The transverse stripes of the muscular fibers of the *heart* are replaced by fat, a form of change seen in the muscular system generally. If the case has been one of the hemorrhagic type, there will be extravasations of blood in the tubules of the kidney, in the endocardium, the peritoneum, the pleura, the mediastinum, and many other places.

Hill [1] has reported a case having points of interest. A girl of fifteen smeared rat-poison on her hands and face for a luminous effect at a "dark séance." She had headache, faintness, bloody vomit, jaundice, constipation, offensive breath, weak pulse, tender abdomen, diminished liver dulness, and coma, dying on the eighth day. At the autopsy the heart was friable and yellow; the gastric rugæ were black, swollen, and softened; the intestines were filled with altered blood, and the mucous membrane was altered like that of the stomach; the liver weighed but 45½ ounces, showed a chrome-yellow color externally and internally, was soft, friable, and greasy, and the section displayed lobules marked out by fatty and congested areas. The kidneys showed red pyramids and very pale cortex. The odor of phosphorus was detected in the brain, but there was no luminosity of the organs.

Chronic Poisoning.—Weakly individuals working in match factories, or makers of phosphorus itself, whose occupation requires that they must inhale phosphorus fumes daily, become the subject of "lucifer disease" or "phosphorus necrosis." After several weeks or months obstinate toothache is felt, and when the tooth is extracted, the gum does not heal, but retracts, leaving a suppurating bony surface. Pieces of bone come away, and the disease-process in the marrow and in the periosteum spreads to new areas, other teeth and their sockets become involved, and greater portions of bone necrosed. Accompanying the local mischief, partly caused by it and also aggravating it, is a general disturbance of health characterized by anemia, pallor, weakness, hectic fever, diarrhea, septicemia, purpura, sometimes ending in death by exhaustion. These symptoms may be

[1] *Lancet*, 1890.

prevented by dental inspection of workmen and filling of all carious spots on the teeth, by the circulation of fresh air, by the frequent and systematic use of mouth-washes of sodium bicarbonate, and by the prompt exclusion of any one showing significant symptoms.

The use of "safety-matches" and varieties substituting the red or non-poisonous form is spreading, and with better hygienic measures bids fair to remove this disease from the bills of mortality. Stockman[1] found the bacillus of tuberculosis in the pus formed in the carious jaws, and advocates early operations to remove the original foci at the root of the tooth. He takes the position that in a large proportion of cases the phosphorus, by eroding the bone, makes it susceptible to tuberculous infection, which manifests itself later as miliary tuberculosis, meningitis, or phthisis, pulmonary and abdominal.

Tests.—The tests for phosphorus are its peculiar odor, its luminous appearance in the dark, and the power of reduction possessed by it over silver nitrate.

Detection.—The garlicky odor is suspicious, but may be masked by articles of food having a similar odor, such as onions. If the room be darkened, the breath will shine faintly and phosphorescent spots will be seen upon the lips or clothing. The vomited matters or the urine if put into a test-tube, acidulated with sulphuric acid, and gently heated, will evolve luminous fumes. A piece of white paper molded as a lid to the tube (Fig. 31) should be wet with a drop of a strong solution of silver nitrate. The phosphorus vapor will cause the metallic silver to be reduced as a black spot on the paper. To prove that this is not produced by hydrogen sulphid, the same test should be repeated after adding some lead acetate to fix the hydrogen sulphid in the liquid, or a plug of absorbent cotton wet with lead acetate may be put in the neck of the tube. When the phosphorus is present in minute quantities, it will not be evident by this test unless performed by the careful method of Mitscherlich.

FIG. 31.—Apparatus for testing phosphorus vapor with silver nitrate.

Mitscherlich's Test.—The suspected material is put into a flask (*c*, Fig. 32), and acidulated with sulphuric acid to prevent the escape of ammoniacal vapors. When heated gradually by the sand-bath the phosphorus vaporizes, and is conducted by a long delivery tube to a glass Liebig's condenser, *d*, kept cold by water circulating around the inner tube. The room being totally dark, flashes of light and shining clouds appear in the inner tube at the point where the phosphorus vapors are condensed by their cold surroundings. The odor of the distillate is alliaceous.

[1] *Brit. Med. Jour.*, January 7, 1899.

The tube being vertical, the condensed phosphorus will pass down into a receiver, *e*, where it may be converted to phosphoric acid by the action of nitric acid. The phosphoric acid precipitated by magnesian mixture, collected, ignited, and weighed, will determine the quantity of phosphorus.

If no luminosity has been observed after distilling one-third of the material, the remainder may be subjected to a more searching test. The end of the exit tube of the flask should be detached from the condenser at *d*, and immersed in a solution of silver nitrate. The contents of the flask, *c*, are again heated, while a continuous current of carbon dioxid from the generator, *a*, passes through, slowly carrying the phosphorus unoxidized into the silver nitrate, precipitating black silver phosphid, and leaving some phosphoric acid in solution. Should no black deposit

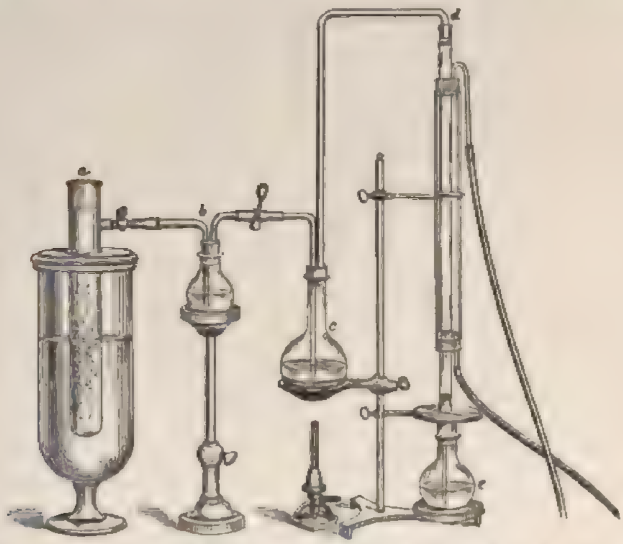


FIG. 32.—Mitscherlich's test for phosphorus: *a*, Generator for CO_2. *b*, wash bottle; *c*, suspected material; *d*, condenser; *e*, receiver for distillate.

appear, the phosphorus may be assumed to be absent. The silver phosphid collected on a filter and washed is suspended in water, and introduced into the hydrogen apparatus employed in the phosphine test described below. The greenish flame is seen even when the quantity is very minute.

Fallacies.—Deductions based upon the detection of phosphoric acid in the distillate when luminosity and free phosphorus have not been obtained may be erroneous. The phosphoric acid may have been brought over by mechanical action.

Interferences.—It can be performed in an organic mixture, but not in the presence of certain chemicals, such as iodin, calomel, and corrosive sublimate. The light will not show in the vapor of turpentine, which may have been given as an antidote. It is not perceived should

alcoholic or ethereal vapors arise from the same mixture. Ammonia, chlorin, hydrogen sulphid, sulphur dioxid, petroleum, creasote, and most essential oils interfere with the phosphorescence.

Delicacy.—This test is extremely sensitive, having yielded unmistakable evidence from one-fiftieth of a grain of phosphorus diffused in three ounces of fluid (Wormley[1]) (1 : 200,000) (Fresenius[2]).

The Phosphine Test.—Having set up the usual hydrogen-generating apparatus,—*i. e.*, flask, pure zinc, and dilute sulphuric acid,—the gas is delivered by a three-way tube, having a side jet, to a wash flask containing the suspected organic mixture, and gently heated. The nascent hydrogen acting on the phosphorus, phosphids, or its lower oxids in the mixture will form phosphine (PH_3), a gas which will escape from the heated flask by a tube drawn out to a jet and having a platinum tip. When lighted, the phosphine, if not too concentrated, will burn with a characteristic green color. It may be contrasted with the flame from the side jet, which should be the pale-blue hue of pure hydrogen. If this side jet is greenish, there must have been some phosphorus in the zinc of the generator. To make sure, the greenish flame should be studied with the spectroscope. If due to phosphorus, it will show one

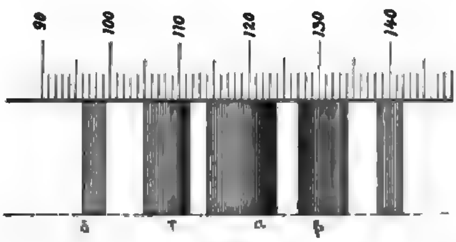


FIG. 33.—The bands represent the green lines of the spectrum of burning phosphine. They are between the lines D and E of the solar spectrum (Boisbaudran).

orange band between *C* and *D*, and several green bands (Fig. 33). Both the color of the flame and its spectrum are best developed if the temperature of the flame is not allowed to rise too high. This may be accomplished conveniently by allowing the flame to impinge against the bottom of a porcelain dish filled with cold water, or by wrapping the burner with a small strip of cloth saturated with cold water.

Phosphorescence in Hydrogen.—This test for free phosphorus only is best performed with the apparatus of Mukerji (Fig. 34),[3] made from a three-necked Woulfe's bottle of one liter capacity, by inserting through close-fitting stoppers a long safety funnel tube (*a*) in one side neck, and a short jet tube (*c*) in the other. Through a loose fitting one at the middle neck rises a tube 11 inches long and one-half inch in diameter, which is closed above by a cork (*b*). From zinc and dilute sulphuric acid in the bottle hydrogen is evolved. Observed in the dark, the gas at the jet should emit no glow, even if commercial chemicals are used. When

[1] Wormley, *Microchemistry of Poisons*, 1885.
[2] Fresenius, *Quantitative Analysis*, American edition, New York, 1886.
[3] *Chem. News*, October 26, 1900.

the chemical action has heated the bottle, the suspected material is introduced through the middle tube or through either neck, quickly closing again with the stopper.

Free phosphorus is vaporized and glows in a sheaf of light at the jet. If the middle cork is removed, the light sinks down through the jet into the bottle, and the glow appears at the outer opening of the middle tube.

Replacing the cork causes the glow to reappear at the jet. If a quantitative estimate is desired, a proper delivery tube may be substituted for the jet and the gas passed into silver nitrate.

FIG. 34.—Phosphorescence in hydrogen.

Special Advantages.—The apparatus is simple, and as no lamp is required for distillation, complete darkness is possible. The amount of air entering by the jet tube is so small in comparison with the quantity of hydrogen continuously evolved that the mixture is never explosive. Before taking apart, the apparatus should be filled with water by the funnel tube.

While this test gives a glow with free phosphorus only, and not with any of its compounds, the *phosphine test* gives a green flame on ignition of the gas when the materials contain phosphorus, phosphids, phosphites, or hypophosphites indifferently. Free phosphorus does not unite with free hydrogen, and the gas here is not phosphine (Roscoe).

Interferences.—Turpentine or ether will prevent the glow in this test. It can be performed in the presence of organic matter, alcohol, iodin, hydrogen sulphid, and many other substances that prevent the glow in Mitscherlich's test.

Delicacy.—Mukerji found the test as sensitive as that of Mitscherlich, getting appreciable effects from 1 : 200,000.

Quantitative Estimation.—Sonnenschein's method for free phosphorus is first to estimate the phosphoric acid by diluting the suspected mixture, filtering a measured fraction, and precipitating with magnesian mixture, estimating as ammonio-magnesian phosphate. Another portion treated on a water-bath with potassium chlorate and hydrochloric acid will have its free phosphorus oxidized to phosphoric acid. This, being estimated, will show an excess over the first portion. The excess is then to be calculated as free phosphorus.

Period for Postmortem Recognition.—Tested by Mitscherlich's method, characteristic phosphorescence has been obtained in putrid organs two months after death and burial. There has been failure, however, to detect the poison even a few days after death, because of

the conversion of the phosphorus into ammonio-magnesian phosphate, or some other salt which has no significance to the medicolegal expert.

SALTS OF THE ALKALINE METALS.

POTASSIUM CHLORATE.

(Chemical formula, $KClO_3$.)

This salt is much used in the manufacture of explosives and flashing powders, and in medicine. In the household it is a common remedy for sore mouth and throat, and through a belief in its harmlessness, often leads to injury. It is familiar as whitish flat crystals with a salty, metallic taste, dissolving freely in water.

Witthaus[1] has collected 89 cases of poisoning from it, 76 of which were fatal; almost all were accidental; 6 were supposed to be suicidal and 3 homicidal.

Symptoms.—If used as a mouth-wash, it is harmless, but when swallowed in large doses it is an irritant, causing abdominal pain, vomiting, diarrhea, and even collapse. Potter[2] has reported a case of poisoning from two teaspoonfuls taken in two days for sore throat. It caused violent intestinal irritation, with black stools, considerable urinary disturbance with black urine, great prostration, and evidences of grave alteration of the blood. A fatal case in a man of thirty-six years has been reported by Ignatieff.[3] Soon after taking a teaspoonful of potassium chlorate he developed cyanosis, renal pain, violent vomiting, jaundice, and bloody urine. A band of methemoglobin was seen when the blood was examined by the spectroscope. He died on the evening of the third day. The autopsy revealed enlargement of liver, spleen, and kidneys. There were interstitial nephritis and capsular hemorrhages of the spleen. Willie[4] observed a case in which large doses had been taken daily for a month with the production of vomiting, diarrhea, dyspnea, weak heart, and a cyanosis due to chocolate-colored change in the blood. Feckler[5] reported a case of a lad of fifteen showing the same symptoms from taking 150 grains in saturated solution within six hours.

When absorbed, it has a peculiar destructive action on the red corpuscles of the blood, converting the hemoglobin into methemoglobin and setting up secondary symptoms, such as jaundice, hemoglobinuria, suppression of urine, bloody tube-casts, delirium, coma, and death as a consequence of the acute nephritis.

Fatal Dose and Period.—Forty-six grains proved a fatal dose in a child three years old. The minimum adult dose reported as fatal is three drams. Dr. Fountain took $1\frac{1}{8}$ ounces with fatal consequences in seven days. If a certain amount is given in divided doses, the effect is

[1] Witthaus and Becker, *Medical Jurisprudence, Forensic Medicine, and Toxicology*, New York, 1896.

[2] *Medical Record*, 1895.

[3] *La Méd. Moderne*, Paris, 1894.

[4] *Brit. Med. Jour.*, 1887.

[5] *Cincinnati Lancet and Clinic*, 1891.

more severe than when given in a single dose. Death has occurred in five hours, but usually it results from nephritis after several days.

Treatment.—Having washed out the stomach with the tube or pump, the secondary effects must be combated with appropriate remedies. The kidney complications will require active local treatment.

Postmortem Appearances.—The marks of gastro-enteritis will be found—*i. e.*, a mucous membrane reddened, thickened, and easily detached. Inflammatory changes are seen in the spleen, liver, and especially in the kidneys. These organs are enlarged and dark-brown in color, from the presence of the altered hemoglobin.

Detection.—As the salt is unchanged in the body it can easily be separated from organic matter by dialysis. Having colored the suspected solution with indigo sulphate and acidulated with dilute sulphuric acid, the addition of sulphurous acid will discharge the blue color if the chlorate be present.

POTASSIUM NITRATE.

(Chemical formula, KNO_3. Synonyms, *Niter; Saltpeter.*)

This salt occurs in large hexagonal prisms permanent in the air. Under the name "Sal Prunelle" it is to be found molded in small balls. It is used as a remedy for cattle; also in the preservation of meat and in the manufacture of gunpowder and other explosives. In the crystalline form it has been taken as a purgative by mistake for magnesium sulphate and sodium phosphate in 8 cases. In 2 cases it has been mistaken for common salt.

Symptoms.—While doses of one dram (4 grams) cause minor degrees of gastric and intestinal irritation, doses of from half an ounce to an ounce excite acute gastro-enteritis. There are vomiting, abdominal pain, diarrhea, perhaps bloody in character, localized muscular spasms, disturbed respiration and heart's action, and collapse.

Fatal Dose.—Though an adult has died from the effects of 2 drams, other cases have recovered from a dose of an ounce.

Fatal Period.—Two hours is the shortest period in which death has taken place; the average duration of fatal cases is somewhat longer.

Treatment.—The stomach must be evacuated by emetics, and the stomach-pump or tube be used to wash out the poison. Bland demulcents may be administered, and the tendency to collapse overcome by stimulants and warm applications.

Detection.—As nitrates are not present in the body, the presence of a notable quantity in the gastric contents or other organic mixture would be significant. By adding sulphuric acid the nitric acid is freed and the tests for that acid can be applied (*q. v.*).

Copper Tests.—Heated in a test-tube with sulphuric acid and copper turnings, nitrates evolve red fumes of nitrogen oxids.

Brucin Test.—Mixed with an equal volume of sulphuric acid, a nitrate solution produces a tint of carmin on the addition of a trace of brucin.

Potassium bitartrate (cream of tartar) and **potassium sulphate** in excessive doses have acted as poisons, inducing abdominal pain, vomiting, purging, great exhaustion, and fatal collapse. There is no specific antidote for either of them. After evacuation of the stomach the inflammatory and depressing symptoms must be treated as they arise.

BORIC ACID AND BORAX.

The local effect of boric acid being very mild, its virtues as a bactericide have led to its use in surgical practice, especially for washing out cavities and sinuses, to prevent septic changes. Cases are recorded of depression, and eruptions of erythema and urticaria following its absorption from wounds and cavities when used too freely.[1] Occasionally graver phenomena have appeared, such as vomiting, diarrhea, bloody urine, and collapse. Fatal results have ensued in a few cases from injecting the solution into abscess-sacs[2] and from washing out the stomach with it.

The toxicology of boric acid and borax is limited practically to the use of these agents as preservatives of food. For meats, they are often mixed with salicylic acid and applied externally. For preserving milk, it is a common practice to add to 1 quart of milk 10 grains of a mixture of equal parts of borax and boric acid. The Agricultural Bureau at Washington, D. C., is at the present time engaged in an extensive series of experiments dealing with this subject, with a view to definite conclusions. These experiments are to be made upon healthy human beings, and are considered necessary because of the conflicting testimony of different observers.

On the one hand, we have the researches of Chittenden[3] and of Liebreich[4] with dogs fed upon articles containing borax and boric acid. To say the least, in both series the digestion of the food was not notably impaired and the animals gained in weight. The same result followed the experiment made by Liebreich upon rabbits and guinea-pigs. No injury appears to have followed the administration of boric acid to pigs, calves, and children by the British Commission.[5] Tunnicliffe[6] made experiments from which he inferred that neither of them affected the health of the children experimented on. Vaughan and Veenboer[7] conclude that in the small amounts required for preserving cream and butter, and that used as an external dust on hams and bacon, both boric acid and borax are unobjectionable from a sanitary standpoint.

On the other hand, the experiments made by H. E. Annette[8] led him to an opposite conclusion. He found boric acid injurious to kittens, and naturally assumed that the use of milk containing it might be hurtful to young infants. Foster and Schlenker[9] found that albumin

[1] *Northwestern Lancet*, 1888, vol. viii., p. 22. [2] *Medical News*, 1882, vol. xl., p. 571.
[3] *American Journal of Physiology*, 1898.
[4] *Vierteljahrsschrift fur gericht. Med.*, 1900; also *Lancet*, January 6, 1900.
[5] *Ibid.*, 1901. [6] *Journal of Hygiene*, 1901.
[7] *American Medicine*, March 13, 1902. [8] *Lancet*, November 11, 1899.
[9] Quoted in report of Kober on Milk Preservatives, United States Senate Commission, 1902.

digestion was impaired by boric acid, which also produced increased desquamation of the intestinal epithelium. Doane and Price[1] made experiments on calves which indicate that borax and boric acid in milk retard digestion to a slight extent.

As these substances are not normal constituents of the body, the wisest course would be to avoid their use until the most conclusive evidence has been adduced that they are free from harm in the amounts as commonly used for preserving food.

Detection of Boric Acid in Meat.—*Jorgensen's test*[2] makes use of the property of neutralized boric acid to take on an acid reaction after treatment with glycerin. The meat is made strongly alkaline with sodium hydroxid, extracted with hot water for several hours, and the extract filtered. The filtrate is evaporated to dryness, incinerated, and the ash dissolved in sulphuric acid. By warming, the carbon dioxid is removed, and on cooling the solution is neutralized by an alkaline hydroxid, using phenolphtalein as indicator.

To 50 c.c. of the neutral fluid 25 c.c. of glycerin are added, and the mixture titrated with decinormal sodium hydroxid solution without regard to the phosphates. The end-reaction is made more definite by the addition of ethyl alcohol.

Detection in Milk.—*Hird's Test.*—Place in a porcelain dish 1 drop of the milk with 2 drops of strong hydrochloric acid and 2 drops of a saturated turmeric tincture. Dry this on a water-bath, cool, and add a drop of ammonia by means of a glass rod. A slaty-blue color changing to green is produced if borax is present. A drop of milk containing $\frac{1}{1000}$ grain of borax will give this reaction.

ALUM.

(Chemical formula, $AlK(SO_4)_2 + 12H_2O$. Synonym, *Alumen.*)

Excessive doses of this salt have produced irritant symptoms sometimes ending in death. Kramolik[3] reported the case of a young man, aged thirty years, who by mistake drank a mouthful of a 10 per cent. solution of alum. Neither the mouth nor the throat showed any marked reaction to the irritant, but the patient vomited thirty-nine times within the forty-eight hours following. Palpation of the stomach was painful. Mucus was found in the vomitus, mingled with blood, imparting a chocolate color to the mass. The urine was stained by blood, and showed morphologically numerous red blood cells, a few leukocytes, and a few hyaline casts. Traces of albumen were also present. The patient was ill for at least thirteen days. The medicolegal interest in it is practically limited to the question of its action when used as a constituent of certain baking-powders which are consumed by the ton in domestic bread-making. In these powders sodium bicarbonate furnishes gaseous carbon dioxid, which is liberated by the action of the alum present, leaving in the bread sodium sulphate and aluminum hydroxid. The fact

[1] *Bulletin* No. 86, Maryland Agricultural Experimental Station, September, 1902.
[2] *Hygienische Rundschau*, vol. x., p. 748.
[3] *Pester Medicinisch-chirurgische Presse*, 1902.

that many thousands of persons use these powders without any perceptible injury, local or systemic, would indicate either that the aluminum hydroxid escapes solution and absorption or that, if changed to a soluble chlorid by the gastric juice, the amount absorbed must be harmless. At the same time it is proper to note that Siem[1] in his researches found that large doses given to dogs and cats subcutaneously caused paralysis of sensation and motion, with fatty degeneration of the liver and kidneys. The safest view is to hold alum as an unnecessary addition to bread, and certainly of no value as a food. Its presence in any but the smallest amount should be considered proof of adulteration.

Detection.—Having incinerated the organic matter in a platinum dish, the ash should be treated with hydrochloric acid, excess of acid removed by heat, a few drops of nitric acid added, and a final solution in hydrochloric acid boiled and filtered.

This solution is not changed by potassium ferrocyanid or hydrogen sulphid, as are solutions containing the heavy metals. With potassium hydroxid a white precipitate falls, redissolved by excess, whereas an excess of the reagent does not affect the precipitate from a solution of the alkaline earths.

Logwood Test.—The most convenient test for alum in bread is made with a freshly prepared tincture of logwood. This tincture is made by digesting 5 gm. of freshly cut logwood chips with 100 c.c. of alcohol. Having diluted 5 c.c. of the logwood tincture with 90 c.c. of water and added 5 c.c. of saturated solution of ammonium carbonate, the mixture is *immediately* poured over 10 gm. of bread in a glass dish. After five minutes the liquid is poured off, the bread slightly washed, and dried at 100° C. A lavender or dark-blue color denotes that alum is present. Pure bread is at first reddish, fading to a yellow or light brown.

Delicacy.—This test yields a distinct blue with 0.02 per cent. of alum, or 7 grains in a 4-pound loaf.

Fallacy.—Several other mineral adulterants produce a somewhat similar reaction.

THE HALOGENS.

IODIN.

(Chemical symbol, I; Synonym, *Iodum.*)

Elementary iodin is a blue-black solid crystallizing in soft, metallic-looking scales which have an unpleasant taste. At ordinary temperatures it gives off slowly an irritating, invisible vapor. Heated to 220° F. it liquefies and then breaks into a rich, violet-colored vapor. Very sparingly soluble in water, it dissolves freely in alcohol, ether, carbon bisulphid, and an aqueous solution of potassium iodid. The tincture and the liniment are dark-brown fluids used as external applications. By mistake these have been taken internally, causing accidental poisoning, which occurrence, however, is very rare.

Symptoms.—It acts as a powerful irritant upon the stomach and bowels, causing pain in the mouth, throat, and stomach, vomiting and

[1] *Schmidt's Jahrbuch*, vol. ccxi.

purging, extreme thirst, fainting attacks, and collapse. When applied by surgeons freely to absorbing surfaces, it may cause systemic disturbances, such as headache, dizziness, mental trouble, along with the above gastric symptoms brought about indirectly. Its elimination by the kidneys involves that organ in inflammation, which may end in suppression of urine. Bellot[1] reported a case of attempted suicide in a woman who took a half-glass of the tincture. Nausea and burning pain in the throat immediately followed. A quart of water was given, but there was no vomiting for an hour, when a dark, thick fluid was ejected, followed by clear blood. In spite of treatment by milk and starch, in a few hours she had abdominal pains and tenderness, dizziness, and syncope. Entire recovery ensued in a few days. The urine was normal. No iodin could be detected in the urine, saliva, or sweat.

Fatal Dose.—Death has resulted from one fluidram of the tincture, containing less than two grains of the element. Ten or twenty grains of the solid would probably be fatal. Recovery has followed a dose of one fluidounce of the tincture.

Fatal Period.—While death has occurred in twenty-four hours, in cases of poisoning from external application it will be delayed for several days. A boy eleven years of age under the care of Culpepper[2] was poisoned by absorption of iodin from a raw surface extending on both legs from the knees to the feet. On the sixth day he died, having suffered from suppression of urine, hemorrhagic stools, vomiting, and purging. Iodin was found in the vomited matters.

Treatment.—Large drafts of tepid water will assist in evacuating the stomach. The antidote is starch in some form, best given in decoction, such as the clear starch of the laundry or as gruels, boiled rice, or arrow-root, given as long as the vomited matters have a blue color.

Postmortem Appearances.—The morbid changes found are such as attend gastro-intestinal irritation, leading to inflammation and excoriation.

Detection.—By agitating organic matters or an aqueous solution of iodin with carbon bisulphid the iodin is separated, making a violet-colored solution. If the iodin is combined, a very small quantity of chlorin water must be used to liberate it. A decoction of starch which has been allowed to cool gives a dark-blue color, due to the formation of iodid of starch. The yellow stains on the skin and lips are removable by ammonia, which would only deepen the stain of nitric acid.

"Iodism."—Excessive doses of iodids or the persistent use of average doses may induce the symptoms of "iodism." Fatal cases are rare. A typical one was reported by Wolf.[3] The patient was unusually susceptible, owing to the fact that she was suffering from renal disease and cardiac hypertrophy. She took six grains four times in one day. The face swelled, and acne and pemphigus appeared, although no more medicine was taken. In twenty-four hours the eruption involved the mucous membrane of the upper air-passages. On the

[1] *La Méd. Moderne*, Paris, 1893. [2] *Therap. Gazette*, 1888.
[3] *Berlin. klin. Wochenschr.*, 1886.

fourth day there was bloody diarrhea. The facial pemphigus passed into deep ulcers. In eight days she died in collapse.

BROMIN.

(Chemical Symbol, Br; Synonym, *Bromum*.)

This element is a dark, red-brown liquid, which vaporizes in red fumes of an unpleasant odor and highly irritating to the mucous membrane of the nose and air-passages. The saturated solution in water is used as a chemical reagent. Alcohol and ether are the best solvents for it.

Symptoms.—Its vapor when inhaled causes symptoms of violent catarrhal inflammation of the air-passages, with cough, constriction of the chest, and hemoptysis. It acts vigorously as a caustic on organic matter, producing, when swallowed, pain in the mouth, throat, and stomach, with eructation of the peculiar offensive vapor. Its powerful local action may bring on collapse in a few hours.

Fatal Dose and Period.—Very few cases of death have been reported. One was caused by one ounce of bromin. In another fatal case a child of ten took what was calculated to be about two grains of bromin. Fatal collapse has come on within seven hours.

Treatment.—Complete evacuation must be secured by emetics and the stomach-pump. The chemical antidotes are protectives, such as mucilaginous drinks made from starch, arrow-root, barley, rice, flour, or meal.

Postmortem Appearances.—A dark-brown stain marks the point of local action ; the mucous membrane is inflamed, softened, loosened, or even corroded.

Detection.—The element may be identified by its color and odor. If it is present as bromid, the bromin must be freed by adding a little chlorin water. When bromin water is shaken with chloroform, the latter takes up the bromin and separates it in a brownish-yellow layer. Starch-water forms the bromid of starch, which is of a deep yellow color.

"**Bromism.**"—This name has been given to the poisonous effects of long-continued dosing with bromids. The symptoms are the fetid odor of bromin on the breath, mental dulness, nervous depression, muscular weakness, absence of sexual feeling, eruptions of acne, bullæ, and pustules. When pushed to the extreme, the bromids have caused exhaustion and fatal heart failure. Eigner[1] reported a case in a woman nineteen years of age, an epileptic, who, without the sanction of a physician, increased the dose of potassium bromid until she became weak, nervous, wakeful, tremulous in all her movements, and complained of loss of memory, headache, and vertigo. She had fetid breath, coryza, and salivation, became delirious, had lobular pneumonia, and died.

[1] *Wien. med. Presse*, 1887.

CHLORIN.

(Chemical Symbol, Cl; Synonym, *Chlorum.*)

The gas is two and a half times heavier than air, has a greenish-yellow color, a peculiar, irritating smell, and is very energetic chemically. Used in the arts as a bleaching agent and in the household as a disinfectant, it is usually generated from "bleaching salt"—*calx chlorata* of the Pharmacopeia.

Symptoms.—When inhaled in small amounts it causes a suffocative feeling and cough. If taken undiluted, it causes difficult breathing, a painful sense of tightness in the chest, and violent cough with hemorrhage. Indirectly the nerve-centers are involved, producing stupor and even heart failure. A fatal case occurred at Cornell University in Ithaca, in 1894, in an old woman the victim of a practical joke.

Fatal Dose.—Fatal consequences are not apt to occur unless the subject is in delicate health, and the gas is taken with little admixture of air.

Treatment.—Fresh air must be given at once, and the pain relieved by the inhalation of ether. The symptoms of acute bronchitis, narcotism, and enfeebled heart's action must be treated by appropriate remedies.

Detection.—The gas can be recognized by its odor and its bleaching action on moist litmus-paper. As chlorin water it has the same properties, and in addition dissolves gold-foil and yields a white precipitate with silver nitrate, insoluble in nitric acid, but soluble in ammonia.

THE HEAVY METALS.

SILVER.

(Chemical Symbol, Ag; Synonym, *Argentum.*)

Metallic silver is not poisonous, as is often demonstrated by the use of silver wire for sutures and the absence of injurious consequences when silver coins have been swallowed accidentally. Cases of acute poisoning are limited to silver nitrate.

Silver nitrate when pure crystallizes in colorless rhombic plates, freely soluble in water, and having a metallic taste. In medicine an official form, known as *argenti nitras fusa* (lunar caustic), is applied as a superficial escharotic, correcting local diseased states.

Of 7 cases reported[1] due to accidental swallowing of the caustic when applied to the throat for local affections, 5 were in children (one of these was fatal) and 2 in adults.

Symptoms.—The contact of the caustic causes instant pain in the throat and stomach, prompt emesis, and later purging of bloody matters. After absorption takes place nervous symptoms supervene, such as vertigo, spasms, disturbed respiration, and coma.

Chronic Poisoning.—An interesting case due to repeated cauterization has been described by Tolmatcheff.[2] A small granuloma of the foot having been removed, the spot was cauterized with solid "caustic" fifteen times in two months. At a later period fifteen more applications

[1] Witthaus and Becker, *Medical Jurisprudence, Forensic Medicine, and Toxicology*, New York, 1896.

[2] *Brit. Med. Jour.*, 1887.

were made in two and a half months. At this point emaciation began, followed in a few weeks by left-sided hemiplegia, with ecchymoses under the eyelids. The patient's face turned a leaden color, the sclerotics were discolored, many brown-black spots appeared all over the body, and a blue line was seen on the gums. Similar discoloration patches, oval and about the size of apple-pips, were found in 800 silver workers in Berlin.[1] They were attributable to absorption through some abrasion of the hands. The general health was unaffected. Microscopic examination of the patches proved them to be due to deposit of metallic silver in the tissues.

A leaden bluish discoloration of the face and possibly of other parts of the body is sometimes brought on by the medicinal use of small doses of silver nitrate given for a long period. No manner of treatment is of any avail to remove this discoloration.

Fatal Dose.—Death has resulted from 30 grains taken by an adult.

Fatal Period.—In six hours after swallowing a piece of "lunar caustic" a child of fifteen months died in convulsions.

Treatment.—Large drafts of common salt and water will favor vomiting and at the same time be the best antidote, forming insoluble silver chlorid. The stomach-pump may be used if necessary. This treatment can be followed up with a diet of eggs and milk.

Postmortem Appearances.—The local action of the caustic will be seen in stains, at first white, and on exposure to light turning black. These stains are found on the lips, in the mouth, on white clothing, and on the mucous membrane of the digestive tract touched by the poison. Gastro-intestinal inflammation is present.

Tests.—*Hydrochloric acid* and *soluble chlorids* precipitate from soluble salts of silver white silver chlorid, insoluble in nitric acid, but readily soluble in ammonia water.

Potassium iodid gives a yellow precipitate, and *potassium chromate* a blood-red precipitate.

Extraction from Stomach-contents.—Finely divided tissues or gastric contents are digested with ammonia and potassium cyanid. The decanted fluid is treated with excess of hydrochloric acid and the insoluble chlorid separated by decantation; the precipitate is washed on a filter with hot water, dried, and reduced on charcoal to metallic silver.

LEAD.

(Chemical Symbol, Pb; Synonym, *Plumbum*.)

The number of deaths from poisoning in England and Wales by lead reported by the Registrar-General for 1883 to 1887 inclusive was 437.[2] In the city of New York, from 1870 to 1891 inclusive, 113 cases of accidental death from lead-poisoning were reported by the Board of Health in 1892. In spite of this great frequency, lead-poisoning rarely figures in the courts, owing to the fact that most of the cases are due to slow absorption of minute quantities, exposure to which is an incident of certain industries dealing with lead or its compounds. The fatal cases

[1] *Op. cit.*

[2] Stevenson, *Guy's Hosp. Rep.*, 1889.

given above represent but a small fraction of the persons who in that period, from numberless causes, suffered from degrees of chronic poisoning more or less serious, but not ending in death. That acute lead-poisoning does occur to some extent is shown by the statistics collected by Witthaus,[1] who found recorded 23 cases, of which 9 were homicidal and 14 non-homicidal.

Properties.—Metallic lead dissolves freely in nitric acid, sparingly in strong sulphuric acid when hot, but not in dilute or cold sulphuric acid, nor practically in hydrochloric acid. The metal, when imbedded in the tissues as a bullet, exerts no local specific action, being insoluble in the fluids there. While not soluble in pure water, the ordinary water served in plumbers' pipes contains enough free oxygen to oxidize a fresh lead surface, which may then form a soluble bicarbonate by the aid of the carbon dioxid present. A portion of it finally forms a crust of insoluble hydrated oxycarbonate, which prevents further action. While silicates,[2] sulphates, and carbonates tend to prevent the corrosive action of water, nitrites, nitrates, and chlorids increase it ; hence a "hard" water-supply is less dangerous when served in lead pipes than a "soft" or purer article.

Lead acetate ($Pb(C_2H_3O_2)_2 3H_2O$) occurs in white masses of acicular crystals. It is soluble in water, and has a taste at first sweetish, hence the popular name, "sugar of lead," but later the taste is styptic and metallic in character. It is present in pharmaceutic preparations as a pill with opium, a compound suppository with opium, and an ointment. The subacetate ($Pb(C_2H_3O_2)_2 PbO$) is present in *liquor plumbi subacetatis*, in a dilute form in Goulard's water, and in the compound ointment or Goulard cerate.

Lead Carbonate.—The paint known variously as "white lead," "flake white," and "mineral white" is a mixture of lead hydroxid and neutral lead carbonate. It is present in the official ointment of lead carbonate.

Poisonous Salts.—The salt which is of most importance in acute poisoning is lead acetate, while chronic poisoning is most frequently caused by lead carbonate.

The subacetate of lead present in Goulard's extract has very much the same effect as the acetate, but greater in degree, as it contains more lead. Lead chromate (chrome yellow), lead oxids (litharge and red lead), and finely divided metallic lead, while not soluble in water, dissolve in the dilute vegetable acids of food and in the gastric juice, and exert a slowly cumulative poisonous action.

"Ledoyen's disinfectant," containing lead nitrate, and "Turner's yellow," or the oxychlorid,—in fact all the salts of lead,—are poisonous, except perhaps the sulphid and sulphocyanid.[3]

Acute Lead-poisoning.—*Symptoms.*—At first they are such as result from a local irritant, and are less likely to be fatal from a single large

[1] Witthaus and Becker, *Medical Jurisprudence, Forensic Medicine, and Toxicology*, New York, 1896.

[2] Crookes, Odling, and Tidy, *Lancet*, 1886.

[3] Eulenberg, *Gewerbe Hygiene*.

dose than from the same amount taken in fractions at intervals. In a few minutes a metallic taste is perceived, and soon afterward the mouth and throat feel dry and burn. Retching and vomiting may appear in less than half an hour and prove obstinate and persistent. Abdominal pains come on in colicky cramps, relieved by pressure. Usually the bowels are constipated; occasionally the stools are bloody, and at a later date they are dark from lead sulphid. The urine is scanty, the face anxious, the skin dry, the breath fetid, and the tongue coated. While the brain is clear, the involvement of the nervous system is indicated by the headache, the pain and cramps in the legs, and the numbness and local palsies which appear a few hours later. After a few days in some cases a blue line is seen on the gums.

Fatal Dose.—It is not known what single dose of lead acetate would prove fatal. Since recovery has taken place in 3 cases after taking one ounce (28.3 gm.) of the acetate, it would seem that the fatal amount must be greater when that salt is the poison. It is probable that the fatal dose of the carbonate would be somewhat less than that of the acetate, though the course of the symptoms would be slower.

Fatal Period.—While death from the acute form is very rare, 23 cases have been collected. It may occur from prostration as early as the second or third day.

Treatment.—The first indication is the washing-out of the stomach by a tube or pump, using a solution of magnesium or sodium sulphate. In the absence of the tube an emetic dose of alum (a soluble sulphate) would be serviceable. When the stomach is quiet, the remainder of the poison can be neutralized and the bowels evacuated by half an ounce of magnesium sulphate (Epsom salt). To check vomiting and colic the best reliance is on hypodermic injections of morphin and atropin.

Postmortem Appearances.—In the few autopsies which have been held in acute lead-poisoning indications have been found of gastro-intestinal inflammation. When life has been prolonged until systemic symptoms appear, mischief has been found in the liver and kidneys.

Chronic Lead-poisoning (Synonyms, *Plumbism; Saturnine Intoxication*).—Judging by the cases reported in the medical journals, chronic poisoning is of very common occurrence. In the vast majority the lead enters the body by accident, as a result of its use in certain industries; in a certain proportion it is caused by contamination of food and drink. In these cases the amount of lead in each dose is so small as to escape detection, but, owing to its extraordinary cumulative action, in time a sufficient quantity finds lodgement in different organs to produce widespread damage.

1. Injurious Industries.—Operatives in the *metal* are liable to have it introduced by inhalation, by dust-particles getting in the hair, beard, or clothing and indirectly into food and drink, and possibly through the skin. In this way many cases have been caused in plumbers, smelters, type-founders, compositors, shot-makers, file-cutters, lead-foil workers, etc. It is even more common in those who work in the lead salts used for colors, such as color-grinders, white- and red-lead makers,

japanners, enamellers, lapidaries, potters, combers of yarn died with chrome yellow, and workers on the lead plates of electric accumulators.

2. Food Contamination.—As lead is slightly soluble in water containing certain salts and gases (see p. 382), its widespread use for pipes in which beverages are kept standing overnight causes it to be introduced into drinking-water, into ale and beer drawn from the cellar, into seltzer-water kept in siphons. Lead oxid is largely used to make a glaze on pottery. From this it may be dissolved by acid foods, as fruit-jellies, pickles, vinegar, lemon-juice. As a constituent of solder and the alloy used to tin iron, it finds access to canned goods containing acids.[1]

As a substitute for the yellow of egg in making sweet cakes chrome yellow has been used by bakers, with very grave consequences. In this condition there is a feeling of "poor health," the appetite is small, digestion impaired, energies feeble, and there is more or less profound anemia. An epidemic of lead-poisoning in the north of France, involving over 100 persons, was investigated by Bertrand and Ogier,[2] who came to the conclusion that the lead was in the flour, obtained by all the sufferers from the same mill, and that contamination came from the elevator buckets, which were "tinned" with lead.

3. Cosmetics.—Most of the lotions called "hair-renewers" are preparations containing sulphur and lead acetate or calcium plumbite.[3] They do not restore the natural pigment, but cause the precipitation of black lead sulphid in the hair structure, so as to simulate the natural color. The use of "flake white" as a cosmetic has caused every form of chronic lead-poisoning.

Symptoms of Chronic Lead-poisoning.—The following typical cases were reported by the writer.[4] A seamstress, aged twenty-eight years, had been using "flake white" as a cosmetic applied freely for more than two years to the face to conceal freckles. Within two years her skin had become dry, pale, and sallow, her conjunctivæ yellow, her hair had fallen out, she had dyspepsia, debility, and low spirits. She had repeatedly suffered from constipation and colic attributed to indigestion. Headache and vertigo had been at one time an every-day experience, and after a year of minor symptoms she went under treatment for melancholia, the cause being at that time undetermined. While confined she left off the cosmetic, but after a month of tonics her mind was restored and she resumed the pernicious habit. One month before she came under observation her hands had become weak and tremulous, her spirits depressed and irritable, and a double wrist-drop had developed. The extensors of the forearms and interossei muscles of both hands were completely paralyzed, the flexors were slightly affected, and the supinators apparently unharmed. There was characteristic loss of electrocontractility to faradism, though slowly interrupted galvanism elicited some response. The gums had a blue line, the lead cachexia

[1] Wightman, *Lancet*, 1888.
[2] *Brit. Med. Jour.*, 1887.
[3] Chandler, *New York Board of Health*, 1870.
[4] *Report of Kentucky Board of Health*, 1881.

was marked, and the cosmetic had been spread thickly to redeem the ravages made by itself. Her younger sister was seen later suffering from wrist-drop, with blue line on the gums, cachexia, with a history of frequent attacks of pain in the elbow and shoulder, with convulsive seizures that had been treated as epileptic. Lead was present in the urine of both.

The relative frequency of the different symptoms was shown in the Philadelphia cases reported by Stewart. They were caused by eating cakes made yellow with lead chromate.[1] Most of the 64 cases exhibited the ordinary signs of impaired nutrition with the characteristic cachexia. They had the colic with constipation, the joint-pains mistaken for rheumatism, and the blue line on the gums caused by the reaction between lead albuminate in the gum and hydrogen sulphid of decomposed food-particles between the teeth. Lead was found in the urine of all cases examined. All the cases whose history covered several months were emaciated. Well-marked lead cachexia was present in 78.21 per cent., and the remainder were all more or less sallow. There was frequent vomiting in 79.68 per cent. Some colic was experienced by 76.56 per cent., and the peculiar rotating umbilical lead colic in 60.93 per cent. In 73.43 per cent. there were joint-pains, growing worse at night and not inflammatory in character. The parts affected most often and most severely were the flexor surfaces of the knees and ankles. Bilateral wrist-drop from paralysis of the extensors of the forearm occurred in only 2 cases. Minor degrees of nervous and muscular disorder in the forearm occurred in 3 other cases. The most serious symptoms were those involving the brain and spinal cord. Headache, so constant and severe as to indicate deep cranial mischief, was present in 73.47 per cent. Brain disease was present in 23.43 per cent., causing epileptiform convulsions in 17.18 per cent., delirium in 3.12 per cent., melancholic mania in 1, and coma in 1. The blue line on the gums was shown by 89 per cent., and probably by 6 out of the 7 remaining. The mortality was 12.5 per cent. Postmortem examination of the viscera was made in 5 cases, and revealed lead in all. Reese found it in the brain and spinal cord five months after death.

The fatal cases were characterized by eclampsia due to encephalopathy. It has been suggested that the gravity of the nervous phenomena was doubtless due in some degree to the chromium present in the poison.

Lead appears to form some stable combination with the substance of the nervous system, and induce thereby disturbed function, if not local destruction, of some essential part of the great centers, as well as of the peripheral nerves. In a case of fatal lead-poisoning examined by Blyth the cerebrum was found to contain lead equivalent to 1½ grains of sulphate, and the cerebellum about ¼ grain. An optical neuritis may cause visual disturbances, but these are sometimes due to the retinitis secondary to the kidney mischief.

Treatment of Chronic Poisoning.—By careful inquiry the source of the lead may be discovered, and the patient should be guarded against

[1] *Medical News*, 1887.

further exposure to it. In the case of operatives in lead-works, emphasis must be laid upon the necessity of grinding the pigments under water to prevent the fine particles escaping as dust into the air; free ventilation is requisite; the hands, nails, and beard should be washed and brushed carefully before eating, and meals should not be taken inside the factory. A weak lemonade of sulphuric acid is sometimes used as a beverage. Its antidotal power may be reinforced by occasional doses of Epsom salt.

It is well to begin treatment with a dose of Epsom salt as an antidote to any lead present in the alimentary tract; colic will call for morphin and atropin administered hypodermically; joint-pains for local fomentations; paralysis for electricity and massage. The natural process of elimination of lead is deliberate. The investigations of Mann[1] show that it escapes slowly by the urine, and five to ten times as much by the bowels, without the use of any special eliminant. Several special eliminants, notably potassium iodid, were given freely without causing any increase in the amount of lead excreted. Careful quantitative tests proved that a slight increase attended the use of hot baths, general massage, and occasional purgation. These last, combined with open-air exercise and wholesome diet, are the means most to be relied on. If potassium iodid is given, care should be taken that it does not increase the anemia. A remission should be allowed during which iron preparations would be of service.

Postmortem Appearances.—In chronic cases the pathologic changes discovered cannot be called characteristic. Where albuminuria has been present, the kidneys are found hard and contracted, the seat of granular degeneration. When colic has been a conspicuous symptom, a portion of the intestines has been found constricted, with a gray-black discoloration of the mucous lining. When there has been local paralysis with atrophy, the muscles involved have been found wasted and fatty, and changes have been discovered in the large cells in the anterior cornua of the cord and in the peripheral nerve-fibers. The blue line around the gums is highly significant.

Distribution in the Tissues.—In examining the bodies of 2 cases suddenly fatal, Blyth[2] separated from the brain of one an appreciable amount of lead, from the liver an amount equivalent to $\frac{1}{4}$ grain of sulphate, from one kidney about $\frac{1}{13}$ grain. Heubel found in a dog killed by chronic lead-poisoning, in parts per thousand—the bones to contain 0.18 to 0.27; the kidneys, 0.17 to 0.20; liver, 0.10 to 0.33; spinal cord, 0.06 to 0.11; brain, 0.04 to 0.05; muscles, 0.02 to 0.04; intestines, 0.01 to 0.02, and traces were detected in the spleen, blood, and bile.

It is a remarkable fact that lead is frequently found in persons apparently healthy—certainly free from all symptoms of lead-poisoning. A fallacious conclusion may be reached if the contents of the stomach should contain a bit of melted solder from a fruit-can or a shot derived from eating game.

[1] *Brit. Med. Jour.*, 1898. [2] *Lancet*, 1887.

In the absence of characteristic symptoms during life, if the amount of lead separated from the tissues should be small, it should not be regarded as significant of lead-poisoning.

Lead in the Urine.—From a case reported by Dercum[1] which had symptoms so vague as to make the diagnosis of lead-poisoning doubtful, Marshall obtained from 400 c.c. (14 fluidounces) of urine as much as 5.2 mg. (0.08 grain) of metallic lead.

There is reason to believe that lead is not an uncommon constituent of the urine. Putnam[2] made 86 urine analyses for lead in the healthy and the sick, with the result of finding lead present in 48 cases. He made allowance for the fact that most of them were chosen because of their exposure to lead by occupation or otherwise, and concluded that so far as his figures are a guide, in not more than 50 per cent. of the community at large can lead be detected in the urine. He noted that the urines of persons known to be in perfect health were almost all free from lead.

Tests.—*1. Hydrogen Sulphid.*—A stream of this gas passed through a lead solution, neutral, alkaline, or slightly acid, yields a black precipitate of lead sulphid, insoluble in the alkaline hydroxids or the moderately dilute acids. If the amount of metal be very small, the precipitate will be brown. Hot nitric acid converts it into soluble lead nitrate, and free sulphur separates; by continued heat the acid converts the sulphur into sulphuric acid, and this precipitates the lead as lead sulphate. A small amount of lead would remain in solution.

Fallacies.—This reagent gives a like precipitate with several other metals, such as copper and mercury. To distinguish the lead, the sulphid may be dissolved in warm dilute nitric acid, filtered, the filtrate evaporated to dryness to expel any excess of nitric acid, the residue taken up with water, and the clear solution tested, as stated below, with potassium iodid, dilute sulphuric acid, or potassium chromate. If the quantity of the precipitate is large, it can be reduced to metallic lead by the blowpipe or charcoal.

Delicacy.—From a solution containing $\frac{1}{25000}$ grain of lead oxid to 10 grains of water Wormley[3] got a faint brownish tint with perceptible cloudiness.

2. Potassium Iodid.—This reagent gives, with very small amounts of lead, a yellow coloration; with larger amounts, a yellow precipitate of lead iodid soluble in boiling water, from which it deposits on cooling in gold-colored hexagonal scales.

Fallacies.—If the lead is small in amount and has been treated previously with nitric acid, a brownish color will be caused by the iodin freed from the potassium, unless the free nitric acid has been neutralized or driven off by heat. Lead iodid is soluble in potassium hydroxid and in strong hydrochloric acid.

Delicacy.—Wormley[4] states that a very small quantity of the reagent

[1] *Medical News*, 1887. [2] *Trans. Assoc. Amer. Phys.*, 1887.
[3] Wormley, *Microchemistry of Poisons*, 1885.
[4] Wormley, *ibid.*, 1885.

will cause a satisfactory deposit of small plates from a solution of $\frac{1}{2000}$ grain.

3. Sulphuric Acid.—This reagent diluted gives a white crystalline or granular precipitate of lead sulphate, which is favored by the addition of alcohol. The precipitate is soluble in hot strong hydrochloric acid, in ammonium acetate, and in a large excess of potassium hydroxid.

Fallacies.—This reagent will also make a white precipitate with barium and strontium salts, and with fairly strong solutions of calcium compounds. The lead sulphate is characterized, however, by turning black with ammonium sulphid.

4. Potassium Chromate or Bichromate.—Either of these reagents precipitates lead as a yellow amorphous deposit soluble in potassium hydroxid and strong hydrochloric acid, but insoluble in acetic acid. A yellowish precipitate produced by potassium chromate in neutral copper solutions dissolves in acetic acid, and is thus readily distinguished from the lead precipitate.

Detection in Gastric Contents, Tissues, etc.—A method suitable for the urine, feces, gastric contents, or the finely divided viscera is the evaporation of the fluid or the dilution of the solids to the consistence of a gruel, the destruction of organic matter with potassium chlorate and hydrochloric acid (see p. 397), and filtration while hot. While some of the lead is apt to remain as insoluble sulphate on the filter, a considerable quantity in a soluble combination with potassium chlorid passes through. In toxicologic analysis, as a rule, the total amount of lead is not in excess of what will be dissolved. The filtrate may be precipitated with hydrogen sulphid, the precipitate dissolved in warm dilute nitric acid, the solution filtered and evaporated to dryness, the residue redissolved in water, and tested with sulphuric acid or potassium iodid.

Detection in Urine.—The following method for the urine, devised in its present form by E. S. Wood,[1] is very delicate. A quart of urine acidified with acetic acid is evaporated to dryness and fused in a crucible with a little pure niter until it becomes white. When the crucible is cool, dilute hydrochloric acid is added *hot* to extract the residue after ignition. The extract is then filtered, and the filtrate treated with ammonia to alkaline reaction, to precipitate the phosphates and iron. Ammonium sulphid is added at the same time to throw down the lead and iron as sulphids. This deposit is washed three times by decantation with hot water; then water acidified with hydrochloric acid is added, and the whole allowed to stand until the next day. It is then filtered through a small filter and the residue washed. A little pure nitric acid is then added drop by drop to dissolve the lead sulphid left on the filter and carry it through as nitrate. This filtrate is collected in a watch-glass, evaporated to dryness, and the final test made by adding a drop of water and a crystal of potassium iodid. A yellow precipitate denotes lead.

Electrolysis.—To electrolyze the filtrate of the hot decoction with potassium chlorate and hydrochloric acid, it is placed in a glass vessel,

[1] Putnam, *Trans. Assoc. Amer. Phys.*, vol. ii.

with a bottom of parchment-paper. This cell is immersed to the surface level in an outer vessel containing distilled water acidulated with sulphuric acid. In the inner cell is placed the cathode of four Grove's cells in the shape of platinum foil 50 cm. square (2 in. by 4 in.). Beneath the parchment diaphragm, near to it and parallel with the cathode on the opposite side, is placed the anode. In six hours the cathode is removed, washed, dried, and cleaned of its lead with warm dilute nitric acid. After driving off the free nitric acid by heat the lead is precipitated by dilute sulphuric acid and an equal volume of alcohol added. After being set aside for twenty hours, the precipitate is washed free from acid with water containing 12 per cent. of alcohol. Decanted, ignited, and weighed, 100 parts of the sulphate equal 68.319 parts of metallic lead.

Quantitative Determination.—While the electrolytic method is preferable when the amount of lead is small, for large quantities it is better to precipitate the lead dissolved by decoction in hot hydrochloric acid with hydrogen sulphid. The precipitate may be converted into sulphate by treating it first with warm dilute nitric acid, filtrating, evaporating, dissolving in water, and precipitating with sulphuric acid, evaporating, igniting, and weighing as above, calculating 68.319 parts of lead for 100 of the sulphate.

MERCURY.

(Chemical Symbol, Hg; Synonyms, *Quicksilver; Hydrargyrum.*)

This is a liquid which changes to vapor spontaneously at all temperatures above 41° F. (5° C.). If finely triturated, the globules remain separate if the trituration has been done in the presence of some substance which gives a coating, such as fatty matter or a confection.

The metal has been given in the pure state to remove obstruction from the bowels mechanically, with no injurious consequences unless retained for a number of days. Taylor[1] cites a case in which, after retention for nine days, some salivation was produced, all the metal not being expelled until the fourteenth day. The metal is present finely divided and possibly oxidized in gray powder (*Hydrargyrum cum Creta*, U. S. P.), blue-mass (*Massa Hydrargyri*, U. S. P.), blue ointment (*Unguentum Hydrargyri*, U. S. P.). In this condition, and also if inhaled in the state of vapor, the metal is converted by the fluids of the body into active compounds which exhibit all its poisonous effects. Among its poisonous salts are the black mercurous oxid, the red mercuric oxid (red precipitate, yellow precipitate), the yellow mercurous iodid, the red mercuric iodid, mercurammonium chlorid (white precipitate), mercuric nitrate (acid nitrate of mercury), mercuric chlorid (corrosive sublimate).

Calomel (*Mercurous Chlorid*).—A heavy, white, insoluble, tasteless powder that is not considered poisonous. If retained, however, it changes to some more active compound, such as the poisonous mercuric chlorid, and then produces systemic symptoms. It is so extensively

[1] Taylor, A. S., *On Poisons*, 1875.

used that milder toxic effects are not infrequent, owing to these changes in the stomach or in the prescription due to incompatible association. It is probable that most of the few fatal cases reported were brought about by the conversion of the calomel by the fluids of the body into some poisonous salt. One fatal case is reported by Runelberg,[1] in which three hypodermic injections of calomel of $1\frac{1}{2}$ grains each were given in one month. Collapse supervened upon salivation and diarrhea. It is converted into mercuric chlorid by nitrohydrochloric acid and chlorin water, and probably to a slight extent also by hydrochloric acid and alkaline chlorids. It is changed to oxid or reduced by the alkaline bases and carbonates. Prolonged exposure to sunlight changes it to metallic mercury and mercuric chlorid.

Corrosive sublimate ($HgCl_2$; *Mercuric Chlorid*) is usually seen in crystalline masses; it sublimes at 180° F. (82.2° C.), and is deposited

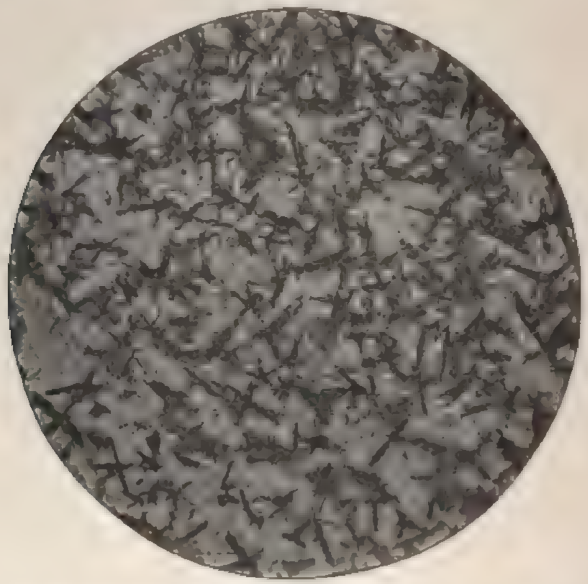


FIG. 35.—Sublimate of mercuric chlorid magnified. Stellate crystals.

in needles, in octahedra, or in stellate aggregations of crystalline plates (Fig. 35). It has no odor, but an acrid, metallic taste. It is soluble in 16 parts of cold water and 3 parts of boiling water, but is far more soluble in solutions of common salt or other alkaline chlorids. It is freely soluble in alcohol and ether, makes a definite insoluble compound with proteid matter, such as albumin, and is fatal to low forms of animal and vegetable life. A solution of it is used in the household to destroy bed-bugs, and by taxidermists to preserve skins and mounted preparations. In antiseptic surgery it is extensively employed as a bactericide in irrigating solutions of 1 : 4000 or even 1 : 1000 of water. Its vogue in

[1] *Arch. f. Dermat. u. Syph.*, 1889.

surgery began about 1880, and its frequency as a poison became increased at the same time. Witthaus[1] has studied 368 records of poisoning collected from the literature of the nineteenth century. Of these, 22 occurred in the decade 1870–79, and 231 between 1879 and 1896. Of the 231 after 1879, 141 were due to irrigations of vagina and uterus, and 48 were fatal. In some death followed surgical operations in which antiseptic irrigation was practised. After the usual symptoms of mercurial poisoning the postmortem appearances attending such poisoning were also discovered. Of the 231, there were 172 which were neither obstetric nor surgical. Of these, 70 were accidental, 47 suicidal, and 27 homicidal.

Symptoms of Acute Poisoning from Corrosive Sublimate.—This salt, however administered, is a very active gastro-intestinal irritant. When taken by the mouth, the symptoms usually begin within a few minutes. The onset is never delayed half an hour. There are an acrid, metallic taste, constriction of the throat, retching, and a burning sensation in the gullet and stomach. A white coating forms at once on the shriveled lining of the mouth, the inflammation of the throat may involve the larynx, and acute swelling of the glottis may cause asphyxia. The pain in the stomach is so severe as to cause fainting. It comes on promptly, attended by nausea and vomiting of material streaked with blood, and later on purging and straining with bloody stools. Free hemorrhages occur from stomach, bowels, or other outlet. The urine is scanty or suppressed, the temperature may be febrile or subnormal, the respiration difficult, the pulse thready and irregular. Death is preceded by collapse, unconsciousness, or convulsions. A typical case was reported by Church:[2] A child, aged three years, by mistake was given a dose equal to 10¾ grains of corrosive sublimate. It immediately caused bloody vomiting, soon followed by intense thirst, drowsiness, weak and rapid pulse, temperature 105.6° F., dilated pupils, and twitching of eyelids. The urine was suppressed, the bowels occasionally moved, with discharges of bloody mucus. There was no salivation, but profuse mucous discharge from the nose. Death occurred in twenty-two hours. Durante[3] has reported the case of a woman aged twenty-five years, who took a large teaspoonful of corrosive sublimate in powder. She vomited, had burning pain in the throat, active diarrhea, suppressed urine, physical depression, stupor, swollen tongue and lips, dysphagia, colic and abdominal tenderness, followed by death in ten days.

Fatal results have followed the application of an alcoholic solution of corrosive sublimate (80 grains to the ounce) to the scalp for ringworm. Andersack[4] records two fatal cases poisoned by the external application of an ointment of corrosive sublimate to cure the itch. In these cases, besides the painful local inflammation, in a few days gastro-intestinal symptoms appeared, such as vomiting and purging with tenesmus. In addition there were stomatitis, fetid breath, fever, scanty

[1] Witthaus and Becker, *Medical Jurisprudence, Forensic Medicine, and Toxicology*, New York, 1896.
[2] *Edinb. Med. Jour.*, 1887.
[3] *Bull. de la Soc. anat.*, 1893.
[4] Blyth, A. W., *Poisons, Effects and Detection.*

urine, and collapse. Sackur[1] has reported a case of fatal acute poisoning from rubbing a small amount of mercurial ointment into cracks of the skin of the hand for a local lymphangitis. In an hour the patient was seized with faintness and vomiting, and on the next day with tenesmus and albuminuria. The later symptoms were continued vomiting, hematemesis, diarrhea with stools of blood, anuria, salivation with gangrenous glossitis and gingivitis, paralysis of the extremities, and death on the sixth day. The lesions were found to be characteristic of mercurial poisoning—*i. e.*, erosions of the small intestine with changes in the large intestine such as attend severe dysentery, and necrotic degeneration of the epithelium in the renal tubules. When the poison is absorbed as a result of irrigation of wounds of the vagina, uterus, or abscess cavities, Butte[2] has found the digestive organs profoundly affected. An earlier effect was serous diarrhea, which afterward became bloody, attended by colic and tenesmus, nausea, and vomiting. He usually found the urine albuminous, containing epithelial cells and granular casts. While there might be severe headache, insomnia, dimness of vision, and transient disturbance of the intellect, the mind was usually clear to the end. The pulse grew weaker, the pupils contracted, the temperature fell, and sometimes an intense erythema appeared. The great frequency of deaths from antiseptic irrigations with corrosive sublimate led Fleischmann[3] to plead for its disuse in obstetric practice. He reported a case in which two vaginal injections were given at the time of examination, the second of which was soon followed by severe abdominal pain, serous diarrhea, and vomiting of bile. The woman was delivered without difficulty, but died in six days after showing characteristic symptoms of mercurial poisoning. The autopsy revealed spongy gums, ulcers of tongue, pharynx, and ascending colon, with acute parenchymatous nephritis.

Fatal Dose.—It is probable that fatal consequences would follow doses of 3 to 5 grains[4] of corrosive sublimate. Recovery has resulted after the administration of 100 grains under prompt treatment by milk, eggs, and emetics. *White precipitate* or mercurammonium chlorid was at one time regarded as non-poisonous. Several deaths from it have been reported—one from 35 grains.[5] *Red precipitate* has caused acute gastro-intestinal irritation when given in doses of two or more drams. *Acid mercuric nitrate*, intended to be used externally only as an escharotic, has been followed by death after such use, and also when administered internally. The *yellow subsulphate*, or turpeth mineral, used in the treatment of croup, has often caused alarming symptoms. Two doses of 3 grains each have been sufficient to cause death.[6]

Fatal Period.—Death may occur in half an hour, but commonly life is prolonged for two to four days, and it may last into the second week.

[1] *Berlin. klin. Wochenschr.*, 1892.
[2] *Brit. Med. Jour.*, 1887.
[3] *Centralbl. f. Gyn.*, 1887.
[4] Taylor, A. S., *On Poisons*, 1875.
[5] Mann, J. D., *Forensic Medicine and Toxicology*, Philadelphia, 1893.
[6] *Med. and Surg. Rep.*, 1884.

Treatment.—Vomiting should be encouraged by large drafts of milk containing emetics. The casein, like all albuminous compounds, acts as an antidote. The most convenient albumin should be given freely. This may be raw eggs, flour paste for its gluten, or blood from a freshly killed fowl, given in milk or water. Magnesia would prove beneficial by conversion of the corrosive sublimate to a less injurious compound. It should not be forgotten that the albuminate of mercury may dissolve in excess of albumin, hence emetics are called for after the antidote has been given. The pain, purging, and tenesmus will require such treatment as is usually given for gastro-enteritis.

Postmortem Appearances (see Plate 4).—Some parts of the alimentary canal are sure to show inflammatory change. In the mouth, throat, and stomach there will be patches of congestion and erosion, or the intestines, especially the colon, may be the seat of inflammation. Eventually the kidneys swell and take on acute inflammation. In Church's case, cited above, the autopsy revealed whiteness of the gums; a band of intense congestion three inches wide along the lesser curvature of the stomach; duodenum normal; mucous membrane of the small intestine of a grayish color, in the lower part greenish; and in the sigmoid flexure was a patch of congestion about the size of a florin. In Durante's case, cited above, the autopsy showed enlarged liver with subcapsular effusions; subpericardial ecchymoses; pale swollen kidneys with small effusions in the pelves; the esophagus red in the upper part; the stomach contained patches of effusion and of softening, with large ulcerations, and the intestines were deep red in limited areas, with ulcers; the brain showed injection of the meningeal vessels.

When death has occurred from absorption of the poison as a result of application to the skin or irrigation of abscesses or of wounds, or of the uterus and vagina, the most important lesions are in the digestive tract. Butte, as cited above, found the inflammation generally limited to the colon. There is hyperemia of the mucous membrane, with easy detachment of the epithelium, patches of superficial necrosis in some parts, and in others a diphtheric coating infiltrating the deeper layers. The kidneys show a characteristic acute parenchymatous nephritis. In some cases the peritoneum is slightly injected. The liver shows no marked lesion, but is generally pale and anemic. The other organs may be unaffected.

Chronic Poisoning or Mercurialism.—The operatives in quicksilver mines, mirror-makers, fine gilders, thermometer- and barometer-makers, furriers, and hatters are liable to a chronic disease ending in paralysis, brought about by the daily introduction and accumulation in the system of minute doses of mercury. Some of the milder symptoms have been induced by the incautious use of mercurials in the treatment of secondary syphilis, and by repeated applications to the skin of a weak lotion of corrosive sublimate for cosmetic purposes.

The symptoms shown in chronic mercurial poisoning are often quite complex. Ptyalism, or salivation, is usually present; the secretion of saliva is profuse, and is attended with swelling and tenderness in the

salivary glands; the gums become red, spongy, and tender, with occasionally a blue line near the teeth; the tongue is swollen and painful; ulcers form in the mouth, and the breath is very fetid. The teeth are loosened, and the alveolar processes sometimes become the seat of acute periostitis. There is usually loss of appetite, with attacks of nausea and vomiting. In some cases colic and diarrhea are present. Soon supervene depressed energies, loss of weight, anemia, and a peculiar cachexia with symptoms of erythema or eczema. The nervous system is eventually involved, showing attacks of mental excitability and insomnia, or perhaps [illegible] of mind. In the end a peculiar fine tremor spreads from the tongue and face to the upper and lower extremities. The tendency of these tremors is to progress from the jerky and intermittent form, brought on by excitement or effort, to the continuous, which persists during sleep. The muscles grow weaker, without [illegible]

Disturbances of sensation are [illegible] neuralgia is a symptom, at times appearing as [illegible] anesthetic patches. Affections of sight and hearing are not infrequent.

[illegible] In [illegible] of the gums. [illegible] A [illegible]

[illegible]

Stomach after Acute Poisoning with Corrosive Sublimate (Von Hofmann).

A hospital nurse poisoned herself with a concentrated solution of corrosive sublimate, and died from collapse in four hours after persistent vomiting. A grayish-white eschar was diffused over the mucous membrane of the lips, mouth, and pharynx. At the necropsy the same appearance was found in the esophagus and stomach. The stomach was firm and contracted, looking as if cooked. The entire mucous membrane and part of the submucosa were colored a uniform pale violet. The walls were thickened, and fell into coarse folds. The changes were those of "coagulation necrosis." The peculiar color is the result of the mouse-gray tint of the coagulated blood in the vessels, blending with the white escharotic tissue of the epithelium.

PLATE 4.

Lith. Anst. v. Reichhold München

Treatment of Chronic Mercurialism.—Improvement usually follows removal of the patient from the surroundings where he was exposed to the poison. Although elimination of a single dose is usually complete in a few days by means of the salivary glands, the kidneys, the intestines, and in less degree by the sweat and milk,[1] still if the period of absorption has been prolonged, as it is in chronic mercurialism, some portion of the poison may be retained, combined with albuminous bodies in an inactive state[2] for many months. To stimulate the process of elimination and to secure the oxidation of the albuminous compound so as to set free the mercury, the bowels should be kept opened, the action of the skin promoted by warm baths, and the best hygienic and tonic regimen instituted. It is customary to administer potassium iodid in small doses in the belief that it changes the deposited poison into mercuric iodid, which is soluble in excess of the potassium salt, and is by this means conveyed into the excretory fluids. It is not easy to reconcile this custom with the results obtained by Souchow,[3] who found that when a patient took potassium iodid with the mercurial the elimination of mercury commenced later, and the quantity eliminated was relatively less than when the mercurial was given alone. As it was also less daily if the iodid was given after the mercurial course, he concluded that the iodid did not facilitate the eliminating process, but rather retarded it, proving worse than useless in chronic mercurialism. For the paralysis, massage and electricity are indicated; for the salivation, mild mouth-washes of potassium chlorate or borax are called for.

Tests.—*1. Sublimation Test for Compounds in the Solid State.*—The suspected solid is first thoroughly dried, mixed with dry sodium carbonate, and heated gently in a reduction tube. A shining ring forms on the inside of the tube in the cooler part. A lens resolves this sublimate into minute shining spheres of metallic mercury. The corresponding sublimate of arsenic and antimony are not of this shape. Rubbed with a glass rod, these globules run together into larger rounded masses. A few scales of iodin left in the closed tube for a few hours will vaporize and convert the mercury into a film of yellow, and later of red, mercuric iodid. If this test is done in a small subliming cell, such as is described under Arsenic (p. 419), and collected on a slide for microscopic examination, it is of very great delicacy.

2. Hydrogen Sulphid Test.—A solution of a mercurial salt acidulated with hydrochloric acid yields a black precipitate when treated with a stream of hydrogen sulphid. The formation of the mercuric sulphid through intermediate stages is shown if the tested solution is strong; the precipitate becomes successively yellowish white, dark yellow, orange, brown, and black. The precipitate is insoluble in caustic alkalis, alkaline sulphids, and nitric or hydrochloric acids. It can be identified by yielding the globular sublimate when dried and heated with sodium carbonate, as directed above. By drying the sulphid in an air-oven and weighing, the quantity of mercury can be calculated.

[1] Bynssen, *Jour. de l'Anat. et de Phys.*, 1872.
[2] Voit, *Physiol.-chem. Untersuch.*, 1867. [3] *Jour. de Pharm. et de Chim.*, 1887.

3. Reinsch's Test.—The procedure is the same as that given under Arsenic, p. 418. A strip of bright pure copper-foil will receive a gray or silvery deposit in a few minutes from a boiling mercurial solution acidified with hydrochloric acid. Having carefully washed the coated copper in water and dried it, the slip should be heated in a small dry reduction tube, and the resulting sublimate examined for globules, as stated above, and tested with free iodin.

Fallacies.—This test yields a metallic deposit on copper from arsenic, antimony, bismuth, silver, and some rarer metals. Coatings of arsenic, antimony, and mercury are the only ones that give a sublimate when heated in a reduction tube.

Mercury is peculiar in its opaque globular form and the bright high lights under reflected light.

Delicacy.—Using capillary reduction tubes of peculiar construction,

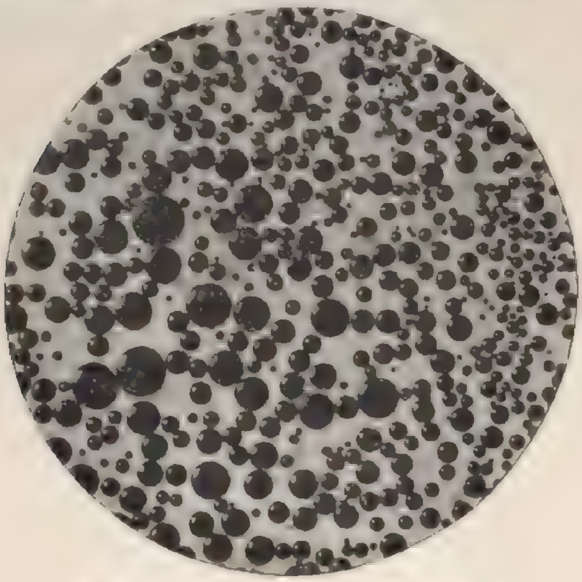


FIG. 36.—Sublimate of metallic mercury magnified.

Wormley[1] has obtained characteristic globules from $\frac{1}{250000}$ grain of corrosive sublimate; under ordinary manipulation $\frac{1}{100000}$ grain is nearer to the limit of delicacy.

4. Galvanic Gold Test.—A band of gold-foil is wrapped about a strip of thin zinc, leaving some zinc exposed, thus making a galvanic couple. Having acidulated the suspected liquid with hydrochloric acid and warmed it, the two metals are hung in it for several hours. A silvery deposit on the gold indicates mercury. After washing the gold successively in water, alcohol, and ether, it may be heated in a reduction tube and the sublimate of mercurial globules produced may be identified, as stated under sublimation test.

[1] Wormley, *Microchemistry of Poisons*, 1885.

5. Potassium Iodid Test.—On adding potassium iodid to a solution of corrosive sublimate or other mercuric salt, a precipitate falls, at first yellow, but rapidly changing to red mercuric iodid. This will dissolve in excess of the potassium iodid.

Distribution in the Tissues.—Riederen[1] gave to a dog in thirty-one days 2.789 gm. of calomel (2.368 gm. Hg). By analysis he recovered 2.2. gm. of mercuric sulphid (1.9 gm. Hg), of which there were in the feces 95 per cent., or 2.1175 gm.; in the urine, 0.055; in the brain, heart, lungs, spleen, pancreas, kidneys, scrotum, and penis, 0.009; in the liver, 0.014; in the muscles, 0.0114. If the poison finds access to the body by external application or by irrigation of other cavities than the alimentary tract, it should be looked for in the liver, the urine, and the kidneys. In the case of acute poisoning from corrosive sublimate reported by Church,[2] cited above, a chemical examination was made of the nasal mucus, the gastric and duodenal contents, the pleural serum, the feces six hours before death, a portion of the liver and one kidney, and not a trace of mercury could be detected. It would appear that in the twenty-two hours which elapsed between the administration of the poison and death total elimination had occurred, though it is possible that the chlorid had been reduced and the metallic globules remained undissolved by the chemical procedures. Other cases have been reported which established the fact that in a few days the whole amount of one poisonous dose given by the mouth may escape from the body.

There is liability to error if the analyst loses sight of the well-known fact to which Taylor[3] has drawn attention—*i. e.*, that traces of mercury are very commonly found in the stomach, bowels, liver, kidneys, and other organs of the cadaver with no history of recent dosage from the poison. These are probably accumulations from small non-poisonous doses of blue-mass or calomel, or perhaps vestiges of a previous mercurial treatment of syphilis.

Detection.—A ready, casual examination can be made of the vomited matters or urine by decanting the liquid portion, evaporating it to dryness, treating with pure hydrochloric acid, and applying Reinsch's test, the galvanic gold test, or the electrolytic test.

Separation from the tissues or other organic matter is accomplished by the systematic method referred to under Arsenic. To disintegrate the organic matter thoroughly, it must be finely minced and heated on a water-bath for some time with equal parts of water and hydrochloric acid, while potassium chlorate is added in small amounts until a clear solution is made.[4] After filtration the solution is heated gently to expel the chlorin, and a stream of hydrogen sulphid is passed until the metal is all precipitated as sulphid. A portion of this sulphid may be tested by reduction and sublimation, or it may be dissolved by gentle heat in nitrohydrochloric acid, the solution evaporated to dryness on a water-

[1] Buchner, *Neues Rep. f. Phar.*, 1868. [2] *Edin. Med. Jour.*, 1887.
[3] Taylor, A. S., *Principles and Practice of Medical Jurisprudence*, 1883.
[4] Consult also p. 324, *et seq.* in section on General Principles of Toxicology.

bath, redissolved in warm water, and the above tests be applied or the mercury separated by electrolysis.

Electrolysis may be performed conveniently by the method of Mann.[1] The suspected solution is put in a glass cell having a bottom of parchment paper, and immersed to a common level in an outer vessel of water acidulated with sulphuric acid. The cathode of a battery of four Grove cells, made of a slip of gold-foil, is fixed into the inner vessel near to and parallel with the bottom. In the outer liquid is set the anode, a strip of platinum opposite to the cathode. After the current has passed six hours, the gold coated with mercury is washed successively with water, alcohol, and ether, and weighed. By heating the gold-foil in a hard glass open tube of known weight, the mercury sublimes and is deposited on the tube.

Quantitative determination may be made by finding the loss of weight of the gold-foil carrying a film of mercury when heated as above described. This gives the weight of mercury in the portion of fluid tested; it can be controlled by calculating the increase of weight in the tube. Instead of using electrolysis, the amount of corrosive chlorid present in any fluid in which mercury is sought may be determined simply by boiling the materials in water, straining, filtering, and agitation of the filtrate with ether, separation, and evaporation of the ethereal extract. The dried residue dissolved in water may be precipitated with volumetric solution of silver nitrate, the chlorin estimated, and from this the weight of mercuric chlorid calculated.

Urine examination may be made by electrolysis, Reinsch's test, or by Mayer's method, which follows: Having evaporated the urine to dryness, the residue, mixed with quicklime and slaked lime, is heated in a combustion tube, condensing the mercury on the cooler part.

COPPER.

(Chemical Symbol, Cu; Synonym, *Cuprum*.)

This element is a heavy reddish metal, which dissolves in nitric acid, in hot sulphuric acid, and, when exposed to the air, is soluble also in hydrochloric acid and in ammonia. Even distilled water will, in time, take up some. One hundred c.c. may dissolve 0.3 mg. of copper or 0.2 grain in a gallon.[2] Natural waters containing salts, especially the chlorids, exert still more solvent powers. The syrups and fats dissolve it, and the fatty acids readily combine with it. Vinegar, acid wines, and subacid fruits kept for a few hours in copper vessels are found to contain the metal.

Distribution in Nature.—Not only is copper to be found in native masses and in its ores,—the carbonates, oxids, and sulphids,—but in minute proportions it is a constituent of many common minerals and soils. Natural water takes up a trace, and vegetation thus derives it from soil and from water. Careful analysis has detected it in edible roots, such as the turnip, in fruits, berries, salads, wheat, barley, and

[1] Mann, J. D., *Forensic Medicine and Toxicology*, Philadelphia, 1893.
[2] Carnelly, *Jour. Chem. Soc.*, 1876.

other cereals, coffee, chocolate, and quinin. From plants as food it is found to be derived by animals—domestic and wild. Even oysters sometimes show a trace. Constantly present in our chief foods, it is not surprising that it is found in the body of man. Leaving out of the count foods possibly contaminated artificially, Blyth[1] estimates that each of us takes daily about 1 mg. (0.015 grain) of copper. While the metal is not to be considered as a poison, its salts, in large doses, act as irritants.

Forms.—The irritant salts are copper sulphate (blue vitriol), copper subacetate (verdigris), and copper aceto-arsenite (Paris green). As the poisonous properties of the last named are dependent chiefly upon the arsenic, it is considered among the compounds of that metal.

Copper sulphate ($CuSO_4 5H_2O$), commonly known under the trivial name of "blue-stone," occurs in large, blue, slightly efflorescent crystals, freely soluble in water, and having a strong metallic taste. It is used in medicine as an external application, for its astringent or mild stimulating qualities. Internally, in doses of ¼ grain to 2 grains, it is given as a tonic and astringent; in doses of 5 to 10 grains it acts as a prompt emetic. It is employed in phosphorus-poisoning as an antidote and also as an emetic. In very large doses it is poisonous, and has been used both for suicidal and for homicidal purposes.

Copper subacetate ($Cu2C_2H_3O_2, CuO$) in an impure form is known as "verdigris." The same name is popularly given to other green salts of copper, as the oleate and carbonate. Verdigris in medicine is used only externally. In the arts it is frequently employed.

Symptoms of Acute Poisoning.—Out of 8 cases registered in England in ten years, 3 were suicidal, 5 were accidental, and none was homicidal. The very disagreeable taste of copper salts prevents the criminal use. The onset of the symptoms may be said to begin with this coppery astringent taste and the feeling of tightness in the throat. In a few minutes nausea and violent vomiting of greenish matters begin. Soon appear thirst, pain in the stomach, and colic, with violent purging of stools having the same green hue of the vomit. Ammonia water added to the green excreta will turn them blue, and thus distinguish this copper-green from bile. The urine is scanty and may become albuminous, inky from changed hemoglobin, and loaded with tube-casts. The later stages are characterized by nervous phenomena, such as pains, spasms which may be tetanic, paralysis, delirium, and collapse. In the course of a few days jaundice appears as a result of involvement of the liver.

Fatal Dose.—Owing to the energetic emetic properties of large doses of copper sulphate, evacuation of the stomach is so prompt that we have no means of determining how much would prove fatal. On the one hand, a child four and a half years old has recovered after a dose of over half an ounce of copper sulphate; on the other hand, an adult has succumbed to a dose of half an ounce of verdigris.

Fatal Period.—As a rule, life is prolonged for several days, the

[1] Blyth, A. W., *Poisons, Effects and Detection.*

patient sometimes almost recovering from the symptoms of gastro-enteric irritation and finally dying from the effects of the absorbed poison. Copper sulphate has caused death in four hours.

Treatment.—Evacuation of the stomach must first be obtained by stimulating the natural effort at vomiting. The antidote is the albumin of egg or the casein of milk. Eggs beaten in warm water should be given freely. If vomiting does not occur or is not active, the stomach-pump should be resorted to and the stomach washed out with milk or eggs and water. A milk diet with castor oil will favor removal from the intestine.

Postmortem Appearances.—Congestion, swelling, softening, and excoriations of the mucous membrane of the stomach and bowels are usually found. The colon sometimes shows large ulcerations. A bluish discoloration of the lining membrane indicates that all the copper has not been evacuated. The liver may be soft and fatty, the kidneys swollen, and the tubules closed with bloody casts. In a case reported by Starr,[1] the blood of the entire body was found coagulated in the vessels and changed to a chocolate color.

Chronic Poisoning.—Until comparatively recent times it was thought that the slow introduction of minute doses of copper was injurious to the tissues by causing such pathologic changes as are known to be due to certain other poisons, such as phosphorus, arsenic, antimony, lead, and mercury. It has been proved by Bernatzic[2] that as a slow poison copper belongs to a different category—that of silver and zinc. To produce toxic phenomena it must be given freely and intentionally. After a long course there are functional disturbances of the muscular and nervous systems, anemia, and cachexia. As soon as the administration ceases the functions are restored and the subject spontaneously recovers from the cachexia. Moulin[3] states that it has not been demonstrated that any doses, however large, which have been taken with food have ever caused death, while medium doses in the beginning act as simple emetics, tolerance is rapidly established, and administration can be continued for six months without danger. Lehmann[4] experimented upon himself and his pupils with the sulphate and acetate, and found that a man could take 75 to 127 mg. (one to two grains) of copper in peas and beans daily, divided in two meals, without effect.

Copper salts are extensively used to impart a lively green color to pickled cucumbers and canned peas and beans. A permanent green compound is formed between copper and an acid derivative of the chlorophyl in the vegetable. Elaborate researches have been carried out in various countries under the highest sanitary authorities to settle the limit of copper admissible as not injurious to health. Moulin[5] thought that there was no medical fact better established than that the amount necessary to give the attractive color is absolutely harmless. This opinion is sustained by the Council of Hygiene, composed of Pasteur, Poggiale,

[1] *Med. Record*, 1882.
[2] *Ency. d. ges. Heilkunde.*
[3] *Jour. de Méd. de Paris*, 1887.
[4] *Munch. med. Wochenschr.*, 1891.
[5] *Op. cit.*

and Brouardel, who reported[1] that "copper in the amounts found in canned goods is not capable of injury to health." By a decision of the New York State Board of Health[2] permission is granted to sell canned peas or beans in the preparation of which copper has been used, provided that the proportion of metallic copper shall not exceed three-fourths of a grain (3 grains of copper sulphate) in a pound avoirdupois. At one time it was generally believed that workers in copper or its compounds, such as malachite, were liable to a disease called "copper colic," which differed from lead colic in that diarrhea was present instead of constipation; there was greater prostration, its duration was shorter, and the prognosis was good. It is now maintained by able investigators that such symptoms are not due to copper, but to the lead and arsenic which are impurities in most ores and in the commercial metal, or to the lead in the solder used by the operators. This is borne out by the fact stated by Chevallier, that after more than one attack "drop-wrist" or lead-palsy is apt to supervene. According to Milton,[3] no symptoms of poisoning are found in certain copper-workers, who show copper as a purplish or bluish line on the gums, whose hair turns green, and whose urine stains the ground green. Tschirch[4] sums up the evidence as follows: "So it appears the contention that there is no chronic poisoning in men or animals is at present uncontradicted."

Tests.—*1. Hydrogen Sulphid Test.*—A stream of hydrogen sulphid passed through an acid solution of a copper salt yields a brownish precipitate of copper sulphid, freely soluble in warm nitric acid, slightly so in excess of ammonium sulphid, but insoluble in the caustic alkalis.

2. Ammonia Test.—A solution of a copper salt is either green or blue. By adding ammonium hydroxid in excess to a slightly colored solution, cupric hydroxid is formed, and dissolved to make a much deeper sapphire-blue solution.

Fallacies.—The salts of nickel give the same deep-blue solution.

Delicacy.[5]—The change in color is recognizable in one grain of a solution containing $\frac{1}{5000}$ grain of copper oxid.

3. Potassium Ferrocyanid Test.—This reagent precipitates from a strong copper solution the reddish-brown copper ferrocyanid. When the solution is very dilute, no precipitate falls, but the solution turns reddish-brown. The brown precipitate is insoluble in acetic and hydrochloric acids, but with ammonium hydroxid forms a greenish-blue liquid.

Fallacies.—Solutions of uranium salts yield a similar brown precipitate, but when this is treated with excess of ammonium hydroxid, the liquid is yellow, not blue.

Interferences.—A trace of iron will give a blue color with this reagent and thus mask the result.

Delicacy.[6]—A distinct red reaction can be obtained from $\frac{1}{25000}$ grain of copper oxid.

4. Iron Test.—This test separates copper in the metallic state. It is

[1] *Annales d'Hyg.*, 1880.
[2] *Med. News*, 1887.
[3] *Buck's Hygiene*, New York.
[4] *Das Kupfer*, Stuttgart, 1898.
[5] Wormley, *Micro-Chemistry of Poisons*, 1885.
[6] *Ibid.*

performed by immersing a steel needle or other piece of bright steel or iron in the suspected liquid slightly acidulated. If copper is in solution, it will be deposited as a reddish layer on the iron. To prove that this film is copper, it is dipped in ammonium hydroxid and exposed to the air, when the film of copper turns blue.

5. *Galvanic Zinc Test.*—Very delicate results can be obtained by immersing in a copper solution a galvanic couple made by wrapping platinum wire around a piece of zinc-foil. The platinum is soon discolored by a deposit the nature of which can be established by exposing it to the vapors arising from potassium bromid when treated with sulphuric acid. The deposit changes in color, and if rubbed on white porcelain, leaves a violet mark.

Delicacy.—According to Blyth,[1] $\frac{1}{100}$ milligram of copper in solution can be recognized by this test.

6. *Electrolytic Test.*—Having obtained the copper in solution and concentrated it, make it acid with hydrochloric acid, and put it in a weighed dish of platinum which is connected with the zinc pole or cathode of a battery. A strip of platinum-foil as anode is immersed in the tested solution for twenty-four hours. In that time all the copper will be deposited on the platinum dish. To make a quantitative estimate, the dish must be washed, dried, and weighed again. The gain represents the total amount of copper in the volume of tested solution.

Separation from Animal Matters.—The organic matter in the contents of the stomach or in the liver, brain, or other tissues, must be destroyed by burning to an ash and extracting with nitric acid, or by boiling with hydrochloric acid and potassium chlorate, according to the systematic procedure given under arsenic.[2] By evaporation, the excess of acid can be removed, and the residue, dissolved in acidulated water, may be tested by the methods given above.

BISMUTH.

(Chemical Symbol, Bi.)

The study of the toxic action of bismuth is practically that of the salt most commonly used in medicine, the *subnitrate*. This is a heavy, white, tasteless, insoluble powder, sometimes used as a cosmetic under the name of "pearl white." It is much used as a local sedative for gastric and intestinal irritation, and is given almost *ad libitum*. At one time most samples were imperfectly freed from the arsenic, which is found associated with bismuth in its ores. Antimony, lead, and a trace of tellurium have been found in it.[3]

Symptoms.—While the salt itself has no taste, yet in cases of poisoning a peculiar metallic taste is complained of, accompanied by salivation, foul breath, and sore mouth. There are vomiting, abdominal pain, and purging of stools, dark from bismuth sulphid. Sometimes a

[1] Blyth, A. W., *Poisons, Effects and Detection.*
[2] Consult also p. 325, in section on General Principles of Toxicology.
[3] *Amer. Druggist*, 1884.

black discoloration appears upon the gums, but may spread over the whole mouth.

The strong garlicky odor of the breath sometimes observed has been attributed to tellurium, which produces this effect, although the amount is very minute. As the gastro-enteric symptoms are similar to those of arsenic, the toxic action of bismuth was at one time ascribed to that impurity. Dreesman[1] cites cases showing that large doses internally, as well as free topical applications of bismuth salts, have caused black urinary sediment, albuminuria, and tube-casts, besides the usual stomatitis, loosened teeth, blue gingival line, diarrhea, and ulceration of the intestines. A man, thirty years old, was treated for a severe burn with local applications twice daily of a 10 per cent. ointment of bismuth subnitrate. Within the first three weeks the urine showed black deposits. During the second three weeks there was stomatitis, with pain on swallowing, loosened teeth, and a bluish-black discoloration on the gums and tongue. As the preparation was free from lead, arsenic, or other contaminant, the symptoms were plainly due to absorbed bismuth.

Two cases have been described by Dalche,[2] which show peculiar effects on the mouth, due to the fact that bismuth is eliminated largely by the saliva. These cases are remarkable because equally large amounts administered by the mouth have been without injurious consequences. In one case an extensive burn was treated with local applications of bismuth subnitrate, proved by analysis to be pure. In two weeks there was a severe inflammation of the mouth and throat, with adherent black exudations; vomiting and diarrhea supervened with albuminuria. Bismuth was detected in the urine and the feces. A few days after the application was discontinued the acute symptoms subsided. The second case, after excision of the knee, had the wound dressed with bismuth subnitrate. In a fortnight there were salivation and stomatitis, with bismuth in the urine.

An experimental research on the lower animals, using a pure salt of bismuth hypodermically, caused death after symptoms like those just described.

Fatal Dose.—The earlier reports as to the fatal dose must be taken with much allowance, owing to the fact that until recent times the bismuth salts almost always contained enough arsenic to cause trouble if the dose was a liberal one. Death has followed a dose of two drams. The period of fatality was the ninth day. A dose three times as large has been recovered from.

Tests.—Hydrogen sulphid yields a black precipitate of bismuth sulphid. If this is dissolved in the smallest possible quantity of hot nitrohydrochloric acid and the resulting solution poured into an excess of water, a copious white precipitate of bismuth oxychlorid is thrown down.

Extraction from the tissues is done by boiling the finely divided matter for two hours in dilute nitric acid, the dissolved material sepa-

[1] *Berlin. klin. Wochenschr.*, 1901, No. 36. [2] *Annales d'Hyg. Pub.*, 1887.

rated by filtration, and the filtrate evaporated to dryness. The undissolved organic matter is destroyed with strong nitric acid and then boiled with dilute nitric acid, filtered, and dried.

A solution of both residues is made in 50 per cent. nitric acid and the above tests are applied.

ARSENIC.

(Chemical Symbol, As; Synonyms, *Arsenum; Arsenicum.*)

Poisoning by some arsenical compound is often resorted to by the secret homicide, and comes under notice of the courts more frequently than any other form. Taking suicides and homicides together, it has caused more deaths than any other poison except opium and its derivatives. During the seventeenth century a strong solution of white arsenic, known as *aqua tophana*, was widely employed by the poisoners of Italy and France, who were convicted only by self-confession. In spite of the fact that modern chemistry finds it the easiest of all poisons to detect, it is still used not only by suicides, but by criminals, many of whom escape punishment for years. Frau van der Linden pursued her nefarious career for fourteen years.[1] In Leyden, between the years 1869 and 1883, she is estimated to have poisoned 70 persons, of whom 24 died, including her father, mother, and son. Within a period of eighteen months Mrs. Robinson, of Somerville, Mass., assisted by a quack doctor who knew something about arsenic, poisoned in succession 5 persons of her own family without exciting suspicion until her sixth victim died. Of 8 deaths of trusting friends laid to her charge, arsenic was found in the cadavers of 6. Mrs. Sherman, of New Haven, Conn., succeeded in escaping suspicion while she killed 3 husbands and 8 other persons of her immediate household with arsenic. The peculiar death of her fourth husband led to her conviction.

An instructive series of cases of wholesale poisoning in Havre was investigated by Brouardel, Pouchet, and others.[2] A commission of four experts appointed to decide if certain premises used as a drug-store were unsanitary, reported that the symptoms of chronic ill health ascribed to the state of the house were in reality due to arsenical poisoning. It was then discovered that a clerk, in the course of two years, without exciting suspicion, had poisoned 15 persons, 3 of them fatally. Those that survived, after running the gauntlet of severe disturbance of health, with lesions of the digestive, cutaneous, and respiratory apparatus, were left more or less completely paralyzed.

Arsenic, or ratsbane, is a favorite poison because it is cheap, can be bought as a vermin-killer at any drug-store in the United States, and, owing to its very feeble taste, can be mixed with the food without the victim recognizing the foreign ingredient. The acute symptoms simulate indigestion or *cholera morbus*, and thus the physician is misled. The general practice of undertakers in America of injecting sodium arsenate or other arsenical preparations into the viscera of a corpse to

[1] *Amer. Jour. Med. Sci.*, October, 1886. [2] *Annales d'Hygiène*, 1880.

prevent decay usually makes an insuperable difficulty in the way of conviction, and knowledge of this fact must often embolden the criminal.

The reports of the Registrar-General of England show 51 deaths from arsenic in England and Wales in five years. Counting the number of poison cases thought worthy of record in the medical journals of the world in 1880–89, arsenic figured in 8.3 per cent., poisonous foods in 8.7 per cent., and lead in 10.5 per cent.[1] It is stated by Witthaus that in the decade 1879–89 there were 12 indictments for murder by poison in 31 counties of New York state, in 6 of which the poison alleged to have been given was arsenic, in 1 it was strychnin, and neither morphin nor phosphorus was mentioned. Analyzing 820 cases, he found 45.2 per cent. homicides, 27.3 suicides, and 27.5 per cent. accidental. In the absence of rigid restrictions upon the sale of arsenical compounds, such as are imposed by other governments, the United States has a bad eminence in this respect. The Massachusetts reports give a list of 114 deaths in ten years in that state alone. Only 9 of these were proved to have been homicidal. There were 35 suicides from arsenic in New York city in one year (1891).

Arsenum or Arsenicum.—Free or elementary arsenic is a steel-black mineral with a metallic appearance, subliming at 450° C. (842° F.), and when burned in air, at 180° C. (356° F.), emitting an odor of garlic. It is an ingredient of some "fly-powders." It is present in commercial zinc, iron, and sulphuric acid. It makes a hard alloy with lead, is used in the manufacture of shot, and is often found in Brittania metal. The insolubility of the element as found in these alloys protects us from poisoning by them. Jenkins[2] found arsenic to be the cause of the poisoning induced by eating sardines that had been put up in a soldered tin box. The liability of tin and solder to contain arsenic leads to the regulation of the French Commission of Hygiene that tin should not contain more arsenic than 0.01 in 100.[3]

In testing, it appears as a black stain on copper in Reinsch's test, and as a brown stain on porcelain and a mirror-like ring on glass tubing in Marsh's test. It oxidizes by exposure to the air, and in that state becomes poisonous. When volatilized by heat, it readily unites with oxygen of the air and forms the poisonous vapor of white arsenic.

Arseniuretted Hydrogen (Chemical Formula, AsH_3; Synonyms, *Arsenic Terhydrid; Arsin; Arsonia*).—This is a gas generated by the action of nascent hydrogen on reducible arsenical compounds. It is colorless, has an odor of garlic, and burns into water and arsenic trioxid: $2AsH_3 + O_6 = As_2O_3 + 3H_2O$.

The flame is bluish-white or livid, and if a cold body, such as porcelain, is put into it, the metallic arsenic is deposited as a brown-black spot. The gas, in its course through a small glass tube heated to redness, decomposes and leaves its metallic element condensed on the colder part of the tube as a mirror-like ring. Arsin has a reducing action upon solutions of silver nitrate, causing a black deposit of the metallic

[1] Kobert, *Lehrbuch d. Intoxikationen.* [2] *Med. Herald*, Louisville, 1882–83.
[3] Pouchet, *Annales d'Hygiène*, Paris, 1890.

silver and liberating arsenous acid. It is the most deadly of the inorganic compounds of arsenic. In addition to the early symptoms,—nausea, shivering, dizziness, and prostration,—in the severe cases more serious effects appear. There may be jaundice, with dark-colored blood, the urine may be bloody and suppressed, and coma supervene, ending in death. In cases that have been studied many of the red blood-cells are destroyed and the blood coloring-matter is permanently altered and dissolved in the serum. The tissues of the heart, liver, kidneys, and other viscera show fatty degeneration.

Arsenic Trioxid (Chemical Formula, As_2O_3; Synonyms, *Arsenious Oxid ; Arsenic ; White Arsenic ; Ratsbane*).—Besides the above common names for arsenic trioxid, it has another, incorrect, but official, *acidum arsenosum.* It can be obtained as minute octahedral crystals, as a smooth, heavy powder, or as irregular masses looking like translucent glass and white porcelain. The white cake is at first amorphous and semitransparent, and is then called *vitreous ;* later, by absorption of moisture, it turns to the white, crystalline, opaque, *porcelain-like* variety which has different solubility. The shops usually dispense it as a heavy white powder, partly amorphous, partly crystalline, prepared from the vitreous variety by grinding. The crystalline "flowers of arsenic" obtained by subliming and condensation are made up of octahedral crystals entirely. Samples of the powder from different packages vary in microscopic appearance, and a specimen may often be identified by the size, luster, and relative proportion of the crystals it contains.[1] The taste is so faint and lacking in distinctness as to be unnoticed when mixed with food. While it is sparingly soluble in water, and less so in liquid foods, such as milk, beer, coffee, it may easily be suspended in thick soups or incorporated with bread as a solid.

It is extremely unlikely that arsenic can be taken into the stomach in solution and afterward revert to the solid form.[2]

Owing to the difference in relative amounts of the two forms present in different samples, it is not possible to state the solubility in precise terms, but usually a fluidounce of cold water will dissolve from one-half to three-quarters of a grain (about 1 : 1000).[3] A permanent solution of sixteen grains to the fluidounce (about 30 : 1000) can be made by boiling water with it for an hour. In spite of the greater weight (specific gravity 3.699), powdered arsenic has the curious property of floating on water as a white film. By adding hydrochloric or nitric acid, or by making the water alkaline with the hydroxids or carbonates of the alkalis, the arsenic readily dissolves without change of color. White arsenic has no odor, but if heated on charcoal, it is reduced to metallic arsenic, which in vapor has an odor of garlic.

"Rough on Rats" is the proprietary name of a widely sold gray powder, which is chiefly white arsenic darkened in color by the admixture either of charcoal or of elementary arsenic.

Arsenic is present also in the "Buffalo Carpet-moth Annihilator," and "Tough on Mice," various "fly papers," and other vermin-killers.

[1] Dana, in the Hayden trial. [2] Taylor, A. S., *On Poisons*, 1875. [3] *Ibid.*

Of 22 cases attributed to the administration of a solution of "fly papers," Witthaus[1] counted 7 that were homicidal, 2 suicidal, 6 accidental, and 7 uncertain.

Physiologic Effects.—In the vast majority of cases the local action of arsenic is pronounced. It does not corrode dead and living tissue alike, as would the corrosive acids and alkalis. Vital irritability is required, or the effect on organic matter will be relatively small. Applied to a part, it irritates so profoundly that the phenomena of inflammation appear at once and make rapid progress to the latest stage of local death. It blisters the skin like a burn, and the mucous surfaces respond with equal promptness to its corroding touch. The wide-spread inflammation of the stomach and bowels accounts for numerous cases of rapid death, but the greater number of fatal cases do not exhibit sufficient local mischief to explain the prostration of nervous energy which ends in death. To account for the fatty degeneration of important organs, such as the heart, liver, and kidneys, the theory of Binz[2] has been broached. According to him, cell-protoplasm yields oxygen to arsenous acid, converting it into arsenic acid, and later it reverses its action, reducing the arsenic acid. These unwonted activities induce the morbid changes referred to.

Medical Uses.—Arsenic is much used in medicine as a general tonic, given either alone or in combination with remedies of the same class. It has a high reputation in the treatment of skin diseases, especially of pemphigus and psoriasis. Certain nervous affections, as chorea and neuralgia, are often benefited by it. It will frequently prove of service in chronic bronchitis and asthma. In certain kinds of indigestion a small dose taken before meals will improve the appetite, stimulate the gastric and intestinal glands, and so help assimilation as to increase the weight and energies. As a remedy for chronic malarial troubles it has been employed with great success, acting more slowly than quinin, but of more value than the alkaloid in some apparently intractable cases.

The mode of administration usually practised in skin diseases and chorea is to give five drops of the *liquor acidi arsenosi* or of *liquor potassii arsenitis* (Fowler's solution), well diluted, after meals, increasing the dose one drop daily until the disease is under control or until the eyelids puff and the bowels move too freely, or faint, darting pains are felt in the abdomen. The dose is then reduced to a safer quantity, and persisted in until the warning returns, when it is again reduced. All this time the arsenic pervades all the tissues and can be found in the urine. Occasionally persons are encountered who have an idiosyncrasy for arsenic. Even the minimum dose will produce unpleasant effects. The writer has known cases that would have diarrhea, slight colic, and conjunctivitis after taking two drops of Fowler's solution thrice daily for three weeks. A remarkable case of this kind was reported by Nicholson.[3] After taking in two days fifteen minims of *liquor arsen-*

[1] Witthaus and Becker, *Medical Jurisprudence, Forensic Medicine, and Toxicology*, 1896.

[2] *Arch. f. exp. Path. u. Pharm.*, 1879, vol. xi.

[3] *Lancet*, 1898.

icalis (B. P.), representing not more than one-sixth of a grain of arsenous acid, in doses of three minims after meals, an attack of diarrhea came on, an erythema appeared which covered the whole body, and the eyes were reddened. As soon as the arsenic was discontinued the symptoms disappeared.

It sometimes happens that the early warnings are ignored and the arsenic persisted in until permanent injury is done. The form of injury is neuritis causing paralysis, local or complete. Brouardel[1] mentions the case of a woman aged twenty-two, under treatment for chronic eczema, who took thirty drops of Fowler's solution daily for two weeks, and then forty drops daily for two weeks and three days more. She stopped the doses altogether as the nerves became affected, and in five weeks she was paralyzed.

Symptoms.—If the poison has been in solution and the stomach is empty, the symptoms may appear in eight minutes. If taken solid and with a meal, they may be delayed for as long as ten hours. The usual interval before the first signs is from half an hour to an hour. If a fatal dose has been taken, the symptoms produced are many and various. Departures from the typical forms are frequent, and no symptoms can be considered as characteristic.

In *acute* poisoning, the patient dying within twenty-four hours, the symptoms usually come on within an hour. They are those of a violent irritant producing local inflammation. Added to these, and sometimes occurring independently, are the phenomena of collapse and coma, due to the profound involvement of the central nervous system.

The most conspicuous signs are: (1) An excruciating pain in the pit of the stomach, aggravated by pressure (this burning pain is sometimes absent); (2) sinking sensations and nausea accompany, or may precede, the pain; (3) dry mouth, sore throat, and urgent thirst are common, but may be absent; (4) persistent and forcible vomiting, a sign of an irritability that cannot support the blandest drinks: after ejecting the food, the stomach throws off a rice-water fluid and, later on, a thick mucus, sometimes brown from bile, or sometimes streaked with blood; (5) purging and straining at stools, which may be fetid and bloody, but are apt at first to be thin and watery, like those of cholera morbus (this purging may be absent or insignificant, and in some cases there is obstinate constipation); (6) the urine may be red, bloody, albuminous, scanty, and even suppressed; (7) a feeble, frequent, and irregular pulse ushers in the other symptoms of collapse, the livid and anxious face, sunken eyes, cold and clammy skin; (8) cramps in the calves of the legs, restlessness, spasms ending in unconsciousness.

A small proportion of the cases are classed as *nervous* or *cerebral* because the central nervous system is prominently affected, while the local irritant symptoms, such as vomiting and purging, are slight or wholly absent. The conspicuous nervous phenomena are great prostration, stupor, convulsions, paralysis, collapse, and death in coma.

[1] *Annales d'Hygiène*, 1874.

Such a case was that of Bolle,[1] to whom was administered, in solution, arsenic afterward estimated to have been not less than 0.388 gm. (6 grains). In about forty-five minutes he had gastric pain and vomiting, and died in three hours. The autopsy revealed no evidence of inflammation in stomach and bowels. From the gastric contents the analyst obtained arsenic trioxid 0.0132 gm. ($\frac{1}{5}$ grain), and from the liver and other viscera, 0.00513 gm. (0.078 grain).

A *subacute* form is one favored by ingenious criminals, who give the poison in small doses repeated at intervals so as to cause death by gradual prostration through stages relatively slow. The symptoms make their onset later and are less violent than those of the typical acute form. Most of the cases are of this variety: sooner or later there will be loss of appetite, fainting sensations, nausea, dry throat, retching, shooting pains referred to the stomach and intestines, and diarrhea. These merge into vomiting, great abdominal tenderness, tenesmus with bloody stools, scanty and albuminous urine, jaundice, eczema or erythema, nervous weakness, feelings of numbness and tingling in the extremities, muscular pains, cramps, paralysis, convulsions, and coma.

Under proper treatment the acute symptoms may subside, and some days or even weeks afterward sequelæ will appear. These are attributable to a chronic inflammation of the peripheral nerves, ending in degeneration of the fibers extending from the periphery toward the center, causing loss of sensibility and paralysis in the hands or feet, which may progress until the muscles waste and give the electric response known as the reaction of degeneration. This consequent paralysis was shown in a case reported by Holst.[2] The poisonous dose was a tablespoonful of Paris green. During the first twenty-four hours there was no disturbance of the digestive organs except vomiting and slight abdominal pains. After eight days there appeared a rapidly progressing weakness in the lower limbs. After six weeks examination showed complete anesthesia and paralysis of the lower extremities, with muscular atrophy and partial loss of sensibility and motion in the upper limbs. The degeneration-reaction was elicited from all four extremities. The knee-jerk had gone, but bladder and rectum were unaffected. Gradual recovery followed the treatment by baths, nourishing diet, and potassium iodid. Like interesting features are to be found in a case reported by Kovacs.[3] A man took at least $2\frac{1}{2}$ drams (9.72 grams) of arsenic trioxid. The acute symptoms of gastro-intestinal irritation were controlled by treatment, but a week later an affection of the peripheral nerves developed. First there appeared edema of both legs, and a week later anesthesia of the feet, shooting pains, and unsteady gait. Anesthesia began at the fingers and invaded the arms. Some muscles in all the extremities wasted and showed fibrillary twitchings. The knee-jerk and the superficial reflexes were lost. In four weeks of treatment the ataxia disappeared and the muscles grew firm, the pains and the paresthesia proving the most intractable symptoms.

[1] Casper, J. L., *Handbuch der gerichtlichen Medicin*. Berlin, 1881.
[2] *London Med. Record*, 1887.
[3] *Wien. med. Wochenschr.*, 1890.

Anomalous Cases.—Emphasis should be placed upon the statement made above, that no symptoms can be considered characteristic. Fatal cases have been reported which presented typical postmortem appearances, and yet during life exhibited no pain, vomiting, or purging, and in which thirst was not marked in degree.

Fatal Dose.—Two grains is the smallest fatal dose of white arsenic yet reported.[1, 2, 3] Suicide was accomplished in the case of a woman who had recently aborted by taking half a fluidounce of Fowler's solution, equal to two grains of arsenic trioxid, in broken doses within four days.[4]

Fährenhorst[5] reported a death in six hours from four and a half grains in solution given by mistake to a boy of four years. His symptoms were spasms of the gullet, retching, vomiting, burning in the pharynx, cutting abdominal pains, cramps, trembling, great prostration, collapse, and death. Two weeks after death the autopsy revealed gastritis, duodenitis, laryngitis, pharyngitis, and congestion of the lungs.

After careful search through the literature of the subject Witthaus[6] states that he has failed to find "any record of the clearly established death of an adult from a dose of less than 2 grams (30 grains) of arsenic in the solid form." This dose, taken by a woman of twenty-seven years,[7] caused characteristic symptoms ending by death in twenty-six hours.

To reach this conclusion one must be very rigid in the interpretation of the term "clearly established," ignore those estimates of dosage not based on exact weight, insist upon the absolute health of the subject at the time of administration, and eliminate cases which show in the autopsy any other lesions than those typical of arsenic. Until these extraordinary conditions are fulfilled we must depend for precision of determination upon experiments on the lower animals. Fortunately, we are not without significant data of this kind. Rouyer and Feltz[8] found that the absorption, by dogs, of 0.0025 gm. per kilo (0.018 grain per pound) sometimes, and of 0.003 gm. per kilo (0.021 grain per pound) always, caused death. Making the usual allowance for body weight, this puts the fatal dose for a man of 150 pounds at 0.17 to 0.20 gm. (2.7 to 3.15 grains). These results coincide so perfectly with the careful inferences made by the best authorities (Huseman, Kobert, and others[9, 10]) that they may well be considered as establishing the fact that 3 grains of absorbed arsenic would probably prove fatal to an average man.

[1] Taylor, *On Poisons*, third edition, 1875.
[2] Wormley, *Microchemistry of Poisons*, 1885.
[3] Mann, *Forensic Medicine and Toxicology*, 1893.
[4] *Prov. Med. and Surg. Jour.*, 1848.
[5] Hoffman, *Lehrbuch der gerichtlichen Medicin*, Vienna, 1880.
[6] Witthaus and Becker, *Medical Jurisprudence, Forensic Medicine, and Toxicology*, 1896.
[7] S. M. Ward, *Therap. Gazette*, Detroit, 1885.
[8] *Gaz. d. hop. Paris méd.*, 1875–76, vol. i., 962.
[9] Husemann, *Handbuch der Toxikologie*, 1862.
[10] Kobert, *Lehrbuch der Intoxikationen*; also Blyth, *Poisons, Effects and Detection*; Falck, *Lehrbuch der prak. Toxikologie*; Guy and Ferrier, *Principles of Forensic Medicine*; Tardieu, *Etude Médico-légale*.

Recovery is possible after much larger quantities, as the symptoms vary according to the bodily condition of the person, the state of the stomach, and the form of the poison. Remarkable recovery may ensue if the poison is taken in lumps,[1] or if vomiting evacuates the stomach before absorption has set in.

Fatal Period.—Taylor[2] states the shortest interval before death as twenty minutes. A large dose may overwhelm the entire nervous system so as to bring about collapse and coma within the hour. The average period is about twenty-four hours. In the subacute cases the fatal termination may not occur for several weeks.

Treatment.—The first indication is to *evacuate the stomach* by administering an emetic mixture of a teaspoonful of mustard and a tablespoonful of salt in a tumbler of warm water. This may be repeated in ten minutes, or a hypodermic injection of apomorphin or an emetic dose of sulphate of zinc can be given. Where criminal poisoning is suspected, tartar emetic should be avoided, as it will make detection of arsenic more difficult. The stomach-tube will prove valuable if the stomach is not full of mixed food, pieces of which would occlude the openings in the tube. Large drafts of hot milk and water will facilitate the washing-out of the poison. At the same time the antidote may be given to make the residua insoluble and inert. For this purpose reliance is placed on teaspoonful doses of dialyzed iron or on the freshly made *moist ferric hydroxid,* to convert the arsenic trioxid into ferric arsenate, which is only very sparingly soluble. In the official preparation, Ferri Oxidum Hydratum Cum Magnesia U. S. P., two antidotes are combined. This may be prepared extemporaneously by diluting half an ounce of *tinctura ferri chloridi* with a tumbler of water and adding magnesia in excess. The whole mixture may be taken without straining and repeated several times. If the ferric hydroxid is prepared by adding ammonia water to ferric sulphate or ferric chlorid, then the gelatinous precipitate should be separated from the excess of ammonia by straining through a handkerchief or piece of cheese-cloth. To clear the intestine a dose of castor oil should be given. In spite of evacuants and antidote it sometimes happens that the poison in the form of a powder adheres unchanged to the folds of the mucous membrane.

Postmortem Appearances.—Putrefactive change is usually retarded when the body is permeated with arsenic. If the dose has been large and life prolonged until absorption could take place, this preservative effect will often keep the viscera free from gases and putrid odors for as long as seventeen months. A smaller dose, especially if rapidly followed by death before general diffusion could occur, would not have the same action. The pathologic changes induced are usually those of gastro-enteritis common to the class of local irritants of the stomach and bowels, and if the patient should survive for a number of hours, the absorbed poison will set up fatty degeneration of the heart, liver, and kidneys.

Mouth, Pharynx, and Esophagus.—The repeated acts of vomiting

[1] H. C. Wood, *Therapeutics*, etc., 1874. [2] Taylor, *On Poisons*, 1875.

Stomach from a Case of Acute Arsenical Poisoning (Von Hofmann).

A woman aged twenty-nine years was poisoned with arsenic. On May 27th she was first taken with vomiting and diarrhea, eventuating in death on June 4th. Though there had been seven days' profuse vomiting and diarrhea, a considerable quantity of arsenic was found in the dead body. She had no systematic medical attention. The necropsy showed a flabby, yellowish, and dilated heart, with cloudy swelling of the muscular fibers. Both the liver and kidneys were the seats of parenchymatous degeneration.

The stomach was moderately distended with a watery, turbid, bloody fluid. The inner wall was markedly swelled, softened, and congested. On the crests of the rugæ were ecchymotic points closely set, and mixed with them were injected vessels. At the middle of the greater curvature were dark red, circumscribed areas of confluent ecchymoses denuded of epithelium. Upon and around these areas was a fine, smooth, bright-yellow deposit. The intestines contained a large quantity of watery, turbid fluid resembling rice-water. The mucous membrane was pale violet in color and minutely injected. Its epithelium was loose and easily separated.

PLATE 5.

bring the poison up from the stomach more or less dissolved and active. Inflammatory change sets in at once, and the upper part of the alimentary tract will present enlarged vessels, reddened patches, and erosions.

Stomach.—The lining membrane of the stomach may be covered with a tough mucus or lymph in which white particles of the poison will be imbedded, or, if Paris green has been taken, there may be patches of a bright-green color. Sometimes the arsenic penetrating the gastric walls as far as the peritoneum has been turned into yellow sulphid by the reaction with hydrogen sulphid of putrefaction (see Plate 5). The mucus itself may be abundant and dark, containing blood. Small dark-red dots of effused blood, looking like flea-bites, may stud the surface of the membrane, itself a paler red, obviously due to diffused inflammation. Upon the prominent folds of the mucous membrane these effusions may run together in well-marked streaks of dark-red color. The inflammation may involve the other coats of the stomach, and all of them be found thickened and corrugated. Occasionally localized gangrene ensues. Rarely does the inflammation progress to ulceration, and still more rarely does the ulcer involve the whole structure, causing perforation.

While some degree of gastric inflammation will nearly always be found, it is important to note that death may occur from the cerebral effects ending in coma, while the mischief done to the stomach may be insignificant.

Intestines.—If death be delayed for several days, the whole length of the intestinal tract may be inflamed, but usually the small intestine, and more frequently the duodenum, alone will be involved. There is diffused redness, with scattered patches of a deeper hue, and the contents may be bloody, or perhaps yellow from the formation of yellow sulphid. Like the stomach, the intestines may show little or no sign of inflammation, even with the arsenic present in considerable amount, the death being due to the effect on the central nervous system.

Changes in remote parts may occur if life be prolonged for a number of hours, and are most conspicuous in the *heart*, *liver*, and *kidneys*. Any or all of these may show fatty degeneration. The heart is the seat of effusions of blood under the endocardium, especially of the left ventricle.

Chronic Poisoning.—Peculiar features are found in chronic poisoning that have given rise to the theory that arsenic is *cumulative*. Careful investigation shows that the poison is not stored up in the tissues for such a length of time as are lead and mercury, though the effects appear to accumulate in force and gravity.

Arsenic is readily diffusible, and, passing to the tissues, abides for a few weeks and then is eliminated. The dose may be considerable, yet if the patient lives for three weeks, the arsenic may have entirely disappeared from the soft tissues, but, as Brouardel and Pouchet found, it may still be detected in the bones. On the other hand, cases are recorded where the poison has been found in the liver and bones after two and even six months. In medical practice it is customary to give Fowler's solution in gradually increasing doses until the limit of tolerance is

reached, denoted by the appearance of puffed eyelids and loose bowels. The dose is then reduced progressively. If the full dose is persisted in, the symptoms of chronic poisoning supervene.

The poison has been known to enter by many avenues—inhaled by the lungs, swallowed in food or as excessive medication, applied to the skin by mistake in cosmetics or in the red dye of socks and gloves. The person falls into "poor health," losing appetite and all desire for exertion. Soon twinges of pain, especially sudden colic, will appear. Complaint is made of "sickness" and faintness. The eyelids puff, the conjunctiva is reddened, and the eyes become very sensitive to light. Such signs of indigestion as occasional vomiting, colic, and chronic diarrhea arise. The color fades from the face, the complexion becoming waxy. The person is said to have a wasting fever. Progressive weakness and loss of weight prevail throughout. The hair becomes dry and may fall out, and the nails are brittle and loose. The skin may exfoliate or show spots of darker hue, with eruptions of eczema or erythema. The mouth may lose patches of mucous membrane, form ulcers, and show the symptoms of salivation. The throat, nose, larynx, and bronchial tubes may be affected with a catarrh, causing cough, bloody expectoration, aphonia, and copious coryza. At a later period the nerve-fibers become inflamed and degeneration of this structure is a consequence. At first the sensory nerves indicate the mischief going on by attacks of numbness and tingling in the extremities, which are followed eventually by total absence of normal sensation, or, it may be, by pain and tenderness. When the motor nerves of the hands and feet are involved, there are loss of power in them and wasting of the affected muscles. Even if the poison is discontinued, the paralysis usually lasts for many months, recovery being very slow and generally incomplete. The paralysis may extend until it is general, and death ensue from failure of the heart due to fatty degeneration. A horny condition of the palms and soles, or keratosis, may be produced by the long-continued use of arsenic as a remedy in chronic psoriasis.[1]

Arsenical Applications.—Deaths have been recorded from applications of arsenic, with homicidal intent, to the rectum[2] and the vagina. It has poisoned when used as a urethral injection.

Haberda has reported a case[3] of suicide by means of arsenic contained in a paper bag inserted into the vagina. Incidentally, he referred to the instances of a person who murdered three of his wives successively by introducing arsenic into the vagina after connection; of a single woman who, finding herself pregnant, attempted to produce abortion by this means, but killed herself thereby; of a prostitute murdered by a man who introduced a quantity of arsenic into the vagina, wrapped up in a knot of horsehair.

In these cases of absorption from mucous surfaces and also when

[1] Hutchinson, *Archives of Surgery*, 1895. Other cases have been reported in the same journal (vol. v.) showing that the corns and other similar growths after years of use of arsenic tend to take on malignant action and become epithelial cancer.

[2] Foderé, *Méd. légale.* [3] *Centralblatt für Gynäkologie; Brit. Med. Jour.*, 1897.

applied to the unbroken skin, as ointment, lotion, or powder, the symptoms are much the same as when taken by the mouth. There is, first, a local inflammation, soon followed by nausea, vomiting, thirst, pain, diarrhea, suppressed urine, and nervous symptoms.

An arsenical ointment applied to the scalp of a child to cure an eruption caused death in ten days, with symptoms of gastro-enteritis. The postmortem appearances were redness and inflammation of the stomach and bowels, and the poison was detected in the gastric contents and the liver. By mistake white arsenic was dispensed for a dusting-powder to the skin, with fatal consequences to 17 children.[1] Arsenical plasters used to remove tumors have had severe systemic effects, and even death has been caused by them.[2] In such cases the poison has been found to be distributed throughout the body.

Arsenic-eating.—In view of the deadly nature of this poison, it is not surprising that toxicologists were for a long time skeptical as to the possibility of creating a tolerance for arsenic. It has been proved indubitably that in Styria, Lower Austria, and India individuals have been found who, by carefully increasing the dose at long intervals, have accustomed themselves to take with impunity what in others would produce poisonous symptoms.

These arsenic-eaters take for twenty or thirty years from one-half a grain to two grains or even more of arsenic trioxid at intervals of once a week or oftener, with the intent to increase their powers of endurance. The persons examined have been robust men who lead an active mountain-climbing life. It is not unlikely that to a high constitutional power of resistance they add an unusual activity of the excretory organs. They appear to be especially liable to sudden death.

Skepticism has given place to credulity, and popular writers have helped to create the impression that there are village communities who indulge themselves in arsenic as others do in tobacco. This is not well founded, nor is there evidence to support the opinion that moderate arsenic-taking is common among Americans who wish to improve their complexion. Among its transient pathologic effects are a clear pallor, sometimes a circumscribed flushing of the cheeks, and a glistening of the eye. These soon pass into a waxy skin and puffy eyelids—anything but pleasing to look upon. Very rarely it happens that a person for whom Fowler's solution has been prescribed will take it of his own volition to restore his health, but a persistence in the habit is soon found to be prejudicial and the dose given up. Physicians consider it doubtful if any considerable number of persons find it compatible with comfort for more than a brief period.

In trials for arsenical poisoning it is the custom for the defense to broach the theory of arsenic-eating to account for the arsenic found in the body. A celebrated instance was the case of Mrs. Maybrick, convicted of poisoning her husband at Liverpool, England, in 1889.[3] The symp-

[1] Woodman and Tidy, *Hand-Book of Forensic Medicine and Toxicology*, 1877.
[2] Tardieu, *Étude Médico-Légale sur l'Empoisonnement*, Paris, 1867.
[3] *Guy's Hosp. Rep.*, 1889.

toms were diagnosed by the physicians two days before death as the result of a succession of arsenic doses given within two weeks. The husband of the accused, after tea on April 27th, had an attack of vomiting. On April 28th the vomiting continued and the lower extremities were stiff. After lunch on May 1st and for three days thereafter he was again sick and retching. A week later the vomiting had not left him, his throat was dry and sore, and his bowels were very loose. A diarrhea with straining at stool was marked on May 8th, worse on May 9th, and death ensued on May 11th.

The postmortem appearances corresponded with the symptoms, and arsenic in small amount was found in the liver and intestines. Among the effects of Mrs. Maybrick were found packages of arsenical rat-poison, white arsenic, and arsenical fly-papers. The list of things about the house found to contain arsenic numbered fourteen. She claimed that she made extracts of the fly-papers for use with the various other forms of arsenic as cosmetics. Having been seen to put something secretly into the bottle of beef-extract used by her husband, found afterward to contain arsenic, she stated in her defense that at his request she put in a white powder he was in the habit of using. A colored servant testified that years before, Mr. Maybrick would put arsenic into his beef-tea. It was shown that the accused was on bad terms with her husband and was carrying on a liaison previous to and at the time of his death. Under the instructions of the court the jury discredited the story of arsenic-habit, found her explanations unsatisfactory, and convicted her of the poisoning.

Tests in the Solid Form.—When undissolved, it is easy to recognize the poison by heating it in a sublimation tube and applying other tests to the deposited vapor.

1. Sublimation Test.—Arsenic trioxid sublimes without fusing at a temperature lower than 218° C. (424° F.), the sublimate under a lens presenting octahedra and modified forms, such as tetrahedra and dodecahedra (Fig. 37, c).

Fallacies.—The octahedral form distinguishes the minute arsenical crystals from other volatile white solids subliming at this temperature, such as corrosive sublimate, calomel, and oxalic acid. While many authorities assert that the sublimate of antimony oxid is always amorphous, according to Wormley, Witthaus, and others, it may sometimes occur as octahedral crystals like arsenic.

2. Reduction Test.—This test is applied to any solid compound of arsenic, including Paris green, the two sulphids, and any arsenite. The dry substance is introduced into a reduction tube, part of which has been drawn out to a small caliber at the bottom. It is covered with six times the quantity of a well-dried mixture of three parts of sodium carbonate to one part of potassium cyanid. Heated gently, some moisture may first appear on the tube. This can be removed with a spiral of filter-paper, a swab of absorbent cotton, or by gently heating the moist glass. When the tube is dry, apply strong heat to the flux and then to the arsenic. The arsenical compound is

reduced to metallic arsenic, which is deposited higher up on the tube as a mirror-like ring, black shading to brown or gray (Fig. 37, *a* and *b*).

Fallacies.—The compounds of antimony yield no mirror with this flux, but the compounds of mercury, cadmium, tellurium, and selenium may. When viewed by a lens, the mercury mirror is found to have a fringe of globules. To distinguish the arsenical mirror, the end of the tube must be broken off and the ring heated with the tube aslant. The air playing over the hot arsenic will oxidize it, and the mirror will be vaporized and appear on the cooler parts of the tube as minute white crystals of arsenic trioxid, octahedral in form (Fig. 37, *c*). If another

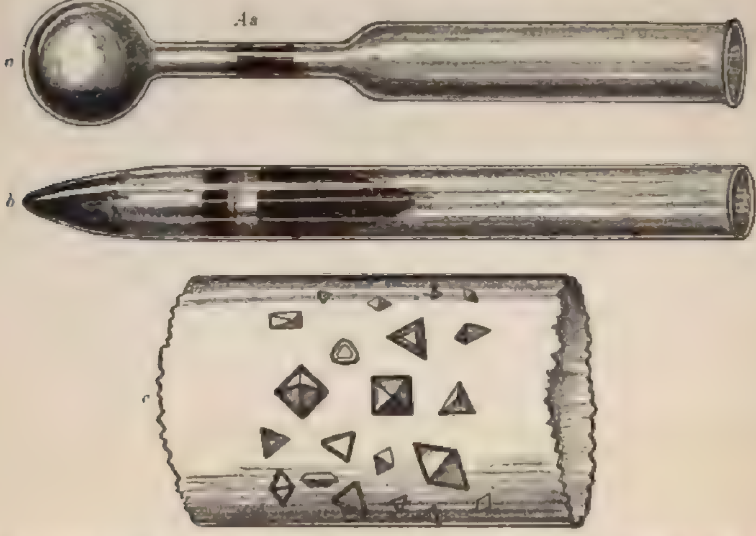


FIG. 37.—*a* and *b* are two reduction tubes showing arsenical mirror after reduction test; *c*, octahedra of As_2O_3 sublimate, magnified.

specimen is treated with a warm solution of chlorinated lime, the mirror will dissolve in a manner characteristic of arsenic.

Delicacy.—If $\frac{1}{1000}$ of a grain of arsenic be tested in a tube contracted to $\frac{1}{20}$ of an inch in diameter, it yields a visible sublimate which will resublime and show many crystals of arsenic trioxid.[1]

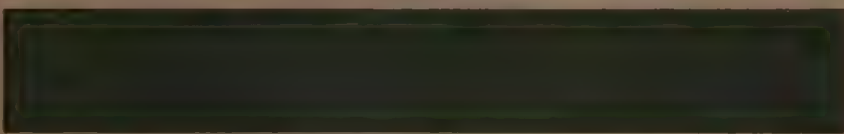
Tests in Simple Solutions.—When the poison has been obtained in solution free from organic or other matter, the following tests will help to identify it:

1. Ammonio-sulphate of Copper Test.—Enough of the reagents for the test can be freshly made by putting about 5 drops of ammonium hydroxid in a test-tube and diluting it with 10 c.c. (3 fluidrams) of water. To this dilute ammonia water a weak solution of copper sulphate is added until the bluish-white precipitate ceases to dissolve. The slight excess of cupric hydroxid should be removed by filtration. The clear blue solution added to a solution of arsenic trioxid will throw down a bright-green precipitate of cupric arsenite—$CuHAsO_3$ (Scheele's

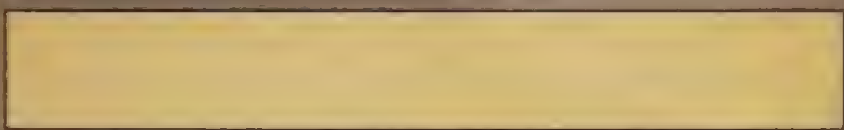
[1] Wormley, *Micro-Chemistry of Poisons*, 1885, p. 259.

PLATE 6.

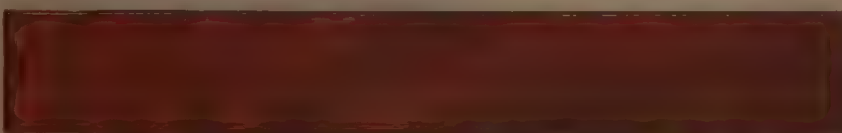
Arsenous sulphid produced in hydrogen sulphid and hydrochloric acid test for arsenic.



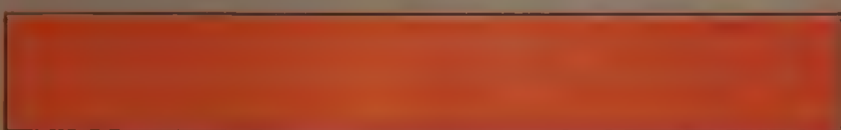
Cupric arsenite produced in ammonio-sulphate of copper test for arsenic.



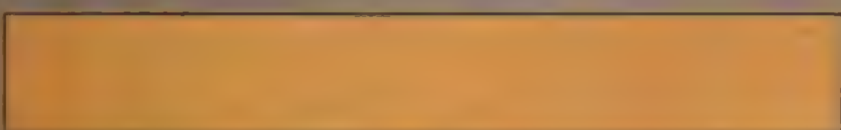
Silver arsenite produced in ammonio-nitrate of silver test for arsenic.



Silver arsenate produced in arsenic solutions by treatment with silver nitrate.



Antimonous sulphid produced in hydrogen sulphid test for antimony.



Stannic sulphid produced in hydrogen sulphid test for stannic compounds.

green) (No. 2, Plate 6). A portion treated with ammonium hydroxid dissolves as a clear blue liquid; another portion will make a colorless solution with nitric acid.

Fallacies.—While no metal but arsenic yields the green precipitate, different organic substances give a green color, and, therefore, interfere with it. The arsenic precipitate, when dried and subjected to the *reduction test*, will give the metallic mirror. Dissolved in hydrochloric acid and subjected to Reinsch's test, the metal deposit will show on copper-foil.

Delicacy.—A green response has been obtained from $\frac{1}{10000}$ of a grain of arsenic.[1]

2. Ammonio-nitrate of Silver Test.—To prepare the reagent, freshly dilute some ammonium hydroxid, as stated in the last test, and add to it a strong solution of silver nitrate until the precipitate of silver oxid formed ceases to dissolve. This reagent yields with solutions of arsenic trioxid a canary-yellow precipitate of silver arsenite, Ag_3AsO_3 (No. 3, Plate 6), which dissolves in ammonium hydroxid and in nitric acid, but not in sodium hydroxid. If dried and heated with flux, as in the reduction test, silver arsenite will be identified by the metallic mirror formed on the cooler part of the tube.

Fallacies.—Other chemicals, such as phosphoric acid, the alkaline iodids, and bromids, will give a like yellow precipitate.

Interferences.—The chlorids, hydrochloric acid, and organic matter decompose the reagent and interfere with this test.

Delicacy.—Minute yellow flakes are yielded by $\frac{1}{10000}$ of a grain.[2]

3. Bettendorff's Test.—A freshly made solution of stannous chlorid is added to the suspected material dissolved in strong hydrochloric acid. Having immersed a small piece of pure tin-foil, the mixture is heated; if arsenic is present, a brown color or a gray-brown precipitate of the metal is formed.

Delicacy.—A brown coloration is yielded by $\frac{1}{10000}$ of a grain, forming $\frac{1}{500000}$ of the hydrochloric acid mixture.

Tests in Complex Solutions.—To detect the arsenic in solutions with other matters the following tests are useful:

1. Hydrogen Sulphid and Hydrochloric Acid Test.—If the solution, acidified with hydrochloric acid and warmed, be subjected to a current of well-washed hydrogen sulphid, bright-yellow arsenic sulphid, As_2S_3 (No. 1, Plate 6), will be thrown down. This deposit is insoluble in cold hydrochloric acid, but hot nitric acid decomposes it and forms solution of arsenic acid. It will dissolve in the alkalis and in ammonium sulphid.

Fallacies.—Yellow or orange precipitates may occur from cadmium, antimony, and tin, and the possible separation of sulphur from the hydrogen sulphid. To verify the nature of the precipitate, it should be separated by filtration, dissolved in ammonia, evaporated to dryness, and subjected to the *reduction test* and the resubliming of the metallic mirror to octahedral crystals.

[1] Wormley, *Microchemistry of Poisons*, 1885, p. 264.
[2] Wormley, *ibid.*, 1885.

Delicacy.—A yellow turbidity, ending in a good deposit, has been obtained from $\frac{1}{10000}$ grain.[1]

2. Gutzeit's Test.—In a test-tube containing 1 c.c. of the suspected solution, either acid or neutral, put about 1 gram of chemically pure zinc and 5 c.c. of a 6 per cent. solution of sulphuric acid. In the upper part of the tube insert a plug of absorbent cotton moistened with lead acetate, and clasp over the mouth of the tube a cap made of three layers of filter-paper. Having wet only the upper layer with a drop of saturated solution of silver nitrate, set aside in a dark box for a time. Arsenic will cause on the paper a bright-yellow spot, which darkens by separation of metallic silver when water is applied to it. The color made by antimony is at no time yellow, but is at once brown or black.

FIG. 38.—Apparatus for Gutzeit's test for arsenic.

3. Reinsch's Test.[2]—The purity of the materials for this test may be established by a blank experiment. A few slips of bright copper-foil should be put into pure water containing one-sixth part of hydrochloric acid, and then heat applied so as to boil for five minutes. The copper remaining bright, the hydrochloric acid may be assumed to be pure; but every detail of this test and others must be paralleled by blank experiments before the analyst can be sure.[3] Having added one-sixth volume of hydrochloric acid to the solution to be tested, a strip of pure copper-foil is put into it and the whole boiled for a few minutes. If arsenic be present as arsenous acid or arsenites, it is deposited as a dark film, purple to steel-gray in color. From *arsenic* acid it is deposited only when the solutions are strong.

Fallacies.—A coating will be left on the copper by arsenic, antimony, mercury, bismuth, gold, and platinum; even prolonged boiling in hydrochloric acid may tarnish it. To verify the arsenical film, the copper slip should be washed, dried with filter-paper, rolled into a cylinder, and inserted into a hard-glass tube open at both ends, all by means of forceps, the finger not touching the foil. When the heat of a spirit-lamp is applied, the metallic film sublimes and is deposited on the tube as a white ring of octahedral crystals of arsenic trioxid, which will dissolve in water and respond to ammonionitrate of silver and the other tests given above.

Besides arsenic, there are two other metals, antimony and mercury,

[1] Wormley, *Microchemistry of Poisons*, 1885.

[2] Consult also section on General Principles of Toxicology, p. 327.

[3] As ordinary copper itself may contain arsenic it should be tested by first giving it a polished surface and then dropping a sample into a boiling mixture of equal parts of solution of chlorid of iron and pure strong hydrochloric acid. If impure, arsenic will show as a black coating.

which make a sublimate under these conditions. Mercury makes a sublimate of shining globules; the antimony sublimate is generally amorphous, but may be in octahedral crystals.

To establish the arsenical nature of the sublimate, the octahedral crystals must be well defined. In order to get the crystals deposited on a glass slide convenient for the use of a quarter-inch objective, the following manipulations will be useful:[1] Having obtained the arsenic stain on copper-foil, the foil is removed, washed, and dried without contact with the fingers, and cut into narrow strips. A subliming tube should be made of thin glass, diameter one-quarter of an inch, length one and three-eighths, sealed at one end, and a lip turned back at the other open end, so that it will hold when hung in an opening made in a sheet of brass four inches by two. The sheet of brass should be laid upon

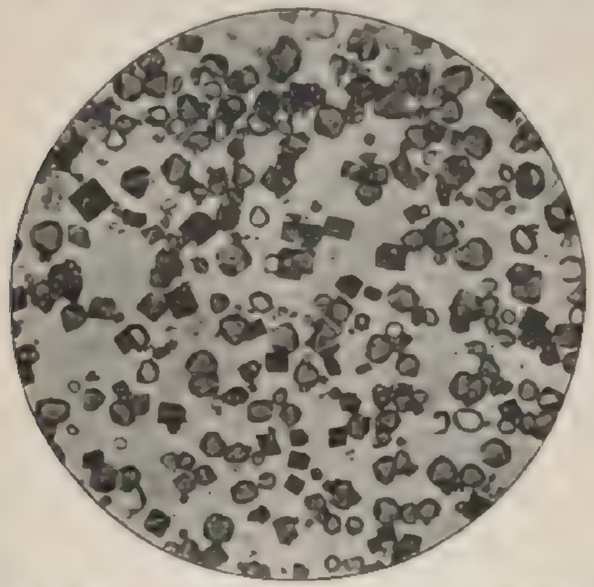


FIG. 39.—Sublimate of arsenic trioxid (magnified 140 diameters).

the ring of a retort-stand, with the tube suspended, and then the tube warmed so as to dry it. After cooling, the tube receives the strips, and a microscope slide, dried by heat, is placed upon it. The glass subliming-tube should then be heated, so as to permit the flame to play also on the bottom of the brass plate. A whitish sublimate will appear on the glass slide in a few seconds, but the heat should not be withdrawn until the white patch begins to clear up at the edges and has a diameter of one-quarter of an inch. The cold slide examined by a one-quarter or one-sixth inch objective will show minute octahedra and tetrahedra, and modifications of these (Fig. 39).

Interferences.—This test does not work properly if nitric acid, a chlorate, manganese dioxid, or other oxidizing agent is present. They cause

[1] *Brit. Med. Jour.*, 1883.

the solution of the copper and prevent the formation of an arsenical coating. With this simple and delicate test it is possible for the physician to make an early diagnosis during life, by examining the vomited matters or urine without any other preparation of the materials than digestion on the water-bath with one part of *pure* hydrochloric acid to six of the tested fluid. The copper slips can then be boiled in this fluid. Any marked darkening of the copper is significant usually of arsenic, antimony, or mercury.

Delicacy.—With ordinary reduction tubes, distinct octahedra form when only $\frac{1}{10000}$ of a grain of arsenic is present; with great care, using very fine tubes, $\frac{1}{50000}$ grain has been revealed.[1]

4. Marsh's Test.—When hydrogen is generated in the presence of compounds of arsenic, they give up the arsenic, which, uniting with it, forms arsenic terhydrid or arsenuretted hydrogen. This is a gas

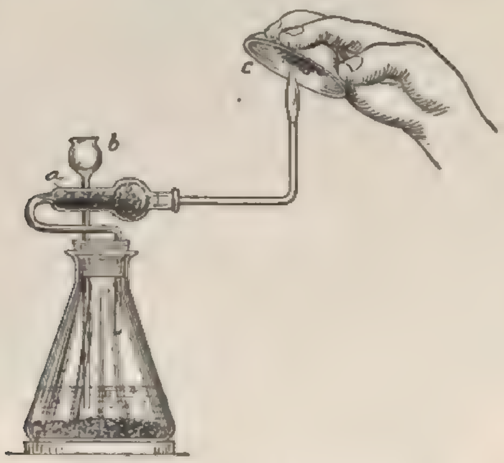


FIG. 40.—Marsh's apparatus for the detection of arsenic.

which, by heat, yields the metallic arsenic for identification by tests already stated. In a flask arranged for generating hydrogen (Fig. 40), with air-tight connections, pure zinc is placed, and pure cold dilute sulphuric acid (one part to six of water) is added to it through the funnel-tube (*b*). The gas is first conducted through a drying-tube containing calcium chlorid (*a*) between plugs of glass wool, and then through an exit-tube of hard glass, about 5 mm. ($\frac{3}{16}$ inch) internal diameter, and 25–50 cm. (10–20 inches) long, which is turned up at the end and drawn out at the tip to make a jet.[2] After waiting a few minutes for the air in the apparatus to escape, a Bunsen flame is applied in the course of the exit-tube, which is heated red-hot, and if no stain appears on the glass after fifteen minutes, the chemicals may be considered pure. The gas-jet should

[1] Wormley, *Microchemistry of Poisons*, 1885.
[2] Chittenden and Donaldson, *Amer. Chem. Jour.*, 1880–81; Fresenius, *Quantitative Analysis*, American edition, New York, 1886.

be ignited, and if arsenical fluid is now poured in by the funnel-tube in small portions, the pale hydrogen jet becomes more luminous and livid in color. If organic matter should cause much frothing, a small quantity of alcohol may be introduced by the funnel-tube.

(*a*) *Marsh's Original Method.*—To prove the presence of arsenic in the gas, Marsh proposed to condense the free metal on cold porcelain held in the flame (Fig. 40, *c*). It is like a spot of soot, black, or seal brown. Many spots can be obtained upon evaporating dishes or crucible lids, and tested later by different reagents to distinguish them from the antimony stains which they resemble closely.

(*b*) *Berzelius' Modification.*—The most delicate, reliable, and, indeed, necessary method for detecting the arsenic with the Marsh apparatus

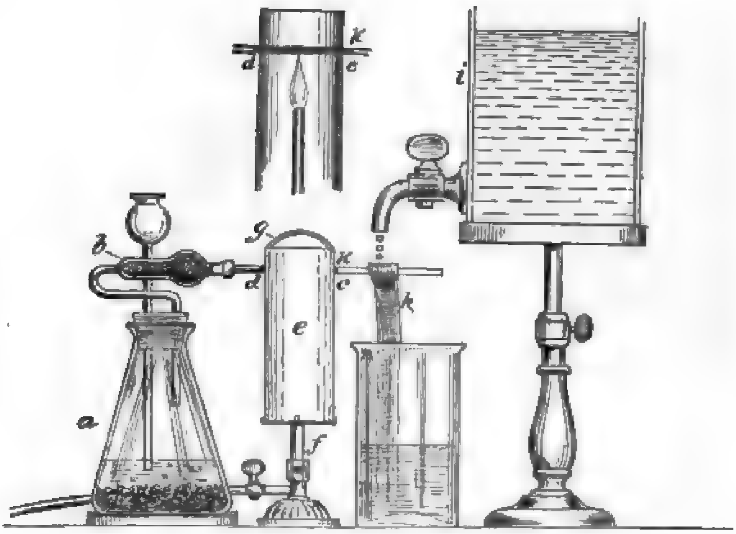


FIG. 41.—Modification of Marsh's apparatus to secure the most delicate results: *a*, Generating bottle; *b*, calcium chlorid tube; *c*, point where hard-glass tube narrows from $\frac{1}{4}$ to $\frac{1}{8}$ inch, a small plug of asbestos inside; *d*, a small plug of asbestos; between *c* and *d* a mixture of dry sodium carbonate and charcoal; *e*, a fire-clay chimney $1\frac{3}{4}$ inches in diameter, with a thin bridge of fire-clay to support the tube between *c* and *d*; *h*, a strip of muslin $\frac{1}{4}$ inch wide wrapped around the tube and tied.

for forensic purposes is to heat the gas while passing through the long exit-tube by applying to it one or more burners with chimneys to confine the heat. For the best results the tube may be constricted at points just beyond the part heated, and the constricted part kept cold by a wet muslin strip (*h*, Fig. 41).

If the heat is maintained for an hour, all the arsenic will be separated from the mixture and collected as a mirror-like ring inside the tube between *k* and the strip of wet muslin. The discrimination tests given below can be used to confirm the arsenical nature of this metallic ring as well as for the spots on porcelain.

Fallacies.—Antimony is deposited under the same conditions as arsenic, and in a form closely resembling it, whether in the spots on

porcelain or the mirror-like ring in the heated tube, but the arsenic mirror is at a little distance beyond the flame and brownish shading to black (*a*, Fig. 42), while the antimony is close to the flame, sometimes on both sides of it, and tin-like in luster.

The arsenic stains are soluble in warm solution of *calx chlorata* or in *liquor sodæ chloratæ*, while the antimony is insoluble, or only very slowly and sparingly soluble. Dissolved by heating gently with a few drops of a solution of ammonium molybdate in nitric acid, the arsenic gives a yellow precipitate, whereas antimony forms none. Another deposit dissolved in nitric acid and dried by cautiously heating leaves a whitish spot which, if arsenical, turns red when touched by a drop of strong solution of silver nitrate (c, Fig. 42); if antimonial, there is no

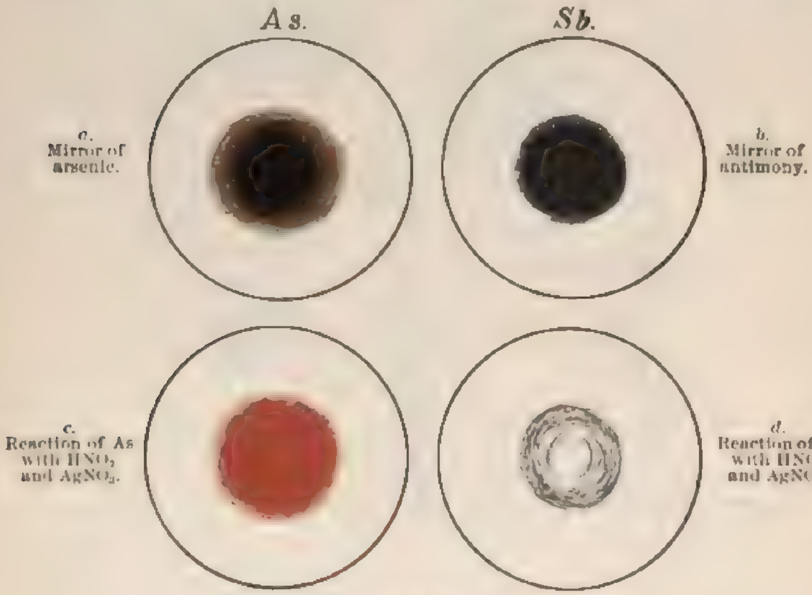


FIG. 42.

change of color (*d*, Fig. 42). Another deposit, if arsenical, dissolves in ammonium sulphid and on evaporation leaves a yellow stain soluble in ammonia, but insoluble in hydrochloric acid. The residue of antimony sulphid would be orange-red, insoluble in ammonia, but soluble in strong hydrochloric acid.

The extraordinary sensitiveness of most of the tests for arsenic requires that the analyst should be very careful that the apparatus is clean and the chemicals are of ascertained purity. In medicolegal analysis it is well to carry on simultaneously a parallel blank examination, similar in every respect but that of containing the suspected matters. Traces of arsenic have been found in zinc, copper, sulphuric acid, hydrochloric acid, even in filter-paper, and in common illuminating gas.

Glass vessels that have been cleaned with shot may have enough left on them to lead to a false conclusion.

Interferences.—The perfection of Marsh's test is impaired if chlorids, hydrochloric acid, nitrous compounds, or nitric acid is present. If salts of silver or mercury are present, they may decompose the arsenic terhydrid in the flask as soon as it is generated.

Delicacy.—According to Wormley,[1] to operate this test even on a small scale requires at least 100 grains of the liquid. The least amount that would yield a satisfactory spot on porcelain is about $\frac{1}{5000}$ of a grain of arsenic trioxid. When the gas is not ignited but heated in the exit-tube so as to get all the free metal at one point, we get a degree of sensitiveness beyond that reached by any other test known to chemistry. By this Berzelius' modification of Marsh's test a characteristic deposit on the glass can be obtained from $\frac{1}{50000}$ of a grain dissolved in 100 grains of liquid.

5. Fleitmann's Test.—When zinc or aluminum is heated with excess of potassium hydroxid or sodium hydroxid in a mixture containing arsenic trioxid or trisulphid, the gas arsenic terhydrid is evolved. The apparatus required is a generating flask with a delivery tube dipping into a 4 per cent. solution of silver nitrate. It is sometimes more convenient to use a test-tube covered with filter-paper wet with silver nitrate, as in Gutzeit's test (Fig. 38). The suspected liquid, made strongly alkaline with pure sodium or potassium hydroxid, is put in the flask or the test-tube with a few pieces of sheet aluminum or pure zinc and gently heated. The arsenic terhydrid reduces the silver as a black precipitate, leaving arsenic trioxid and nitric acid in solution. If the test-tube is used, a black spot appears on the paper cover. By means of this test we can detect arsenic in the presence of antimony, as antimony terhydrid is not evolved by it. It will not detect arsenic as arsenic acid, and as it forms solid hydrid in the flask, holding back one-fifth of the arsenic present,[2] it is not available for quantitative purposes. It is liable to a fallacy from the fact that free hydrogen after some time and phosphin both reduce the silver nitrate, hence the presence of arsenic trioxid in the silver solution must be proved (see p. 425).

Detection in Gastric Contents and Viscera.—The vomited matters should be spread in a thin layer on a large dish and carefully searched for grains of white arsenic, Paris green, or yellow sulphid. For casual or preliminary examination the suspected material may be treated as suggested above by Reinsch's test. The presence of the smallest amount is significant, as arsenic is not a constituent of the body nor should it be present in the food. If any compound of arsenic should be found free in the gastric contents, or if it should be obtained by testing, a specimen of it must be carefully marked and set aside to show in court.

When it is desired to make a *quantitative* estimate, it is necessary to destroy the organic matter. This may be done by mincing the tissue into fine shreds, bruising these in a mortar, heating over a water-bath in

[1] Wormley, *Microchemistry of Poisons*, 1885.
[2] Clark, *Jour. Chem. Soc.*, 1893.

pure dilute hydrochloric acid[1] (best made in the laboratory), and adding from time to time small portions of potassium chlorate until the solids are completely dissolved in a clear yellow fluid and continuing a gentle heat until the odor of chlorin disappears.[2] This fluid is treated with solution of sulphur dioxid to reduce the arsenical compound to the arsenous form, then concentrated on a water-bath and filtered. A slow stream of hydrogen sulphid passed through the filtrate will precipitate the arsenic as arsenous sulphid with other matter. The precipitate is collected on a filter, thoroughly washed, and treated with ammonia water, which dissolves out the arsenous sulphid, leaving various impurities behind. The filtered solution is evaporated to dryness in a porcelain dish, the residue warmed with strong nitric acid until completely oxidized, and solution of sodium hydroxid added in slight excess. The mixture is evaporated to dryness, the residue moistened with pure sulphuric acid, and heated cautiously on a sand-bath until fumes cease to escape. The carbonaceous product is boiled out with acidulated water and the solution filtered. The filtrate should be colorless, and, if the process has been properly executed, contains all the arsenic free from organic matter. As the arsenic generally exists in the solution wholly or in part as arsenic acid, sulphurous acid should be added to reduce it to the arsenous condition, and the mixture gently heated until all excess of the sulphur dioxid has been expelled. A definite part of this solution may be reserved for determining the amount of arsenic by precipitation with hydrogen sulphid, as described below, while the remainder may be subjected to Reinsch's, Marsh's, and other tests to establish fully its arsenical character.

The above method is objected to on account of the liability to lose a small amount of arsenic carried off as vapor by the chlorin. This objection cannot be made to the following method (Taylor's[3]), which is designed to convert all the arsenic into a volatile chlorid and to separate the vapor from organic matter by distillation. The organic material should be cut into fine shreds, well dried in a water-oven, and pulverized in a mortar. The powder stirred in strong, pure hydrochloric acid should then be set aside for twenty-four hours. Having adjusted a flask to the inner tube of a Liebig's condenser connected with a receiver, the acid mixture is poured into the flask and heated over a sand-bath almost to dryness. It is treated a second time with hydrochloric acid and again distilled. Both distillates, representing all the arsenic, should be collected in a receiver containing distilled water. For determining small quantities of arsenic the most delicate method is that employed by Chittenden and Donaldson[4] and by Sanger.[5]

Nitric acid assisted by sulphuric acid is sometimes used to destroy the organic matter and oxidize the arsenic into arsenic acid. In such a

[1] Fresenius, *Quantitative Analysis*, American edition, New York, 1886.

[2] Consult also p. 325 in section on General Principles of Toxicology.

[3] Taylor, A. S., *On Poisons*, 1875.

[4] Chittenden and Donaldson, *Amer. Chem. Jour.*, vol. ii., No. 4.

[5] Sanger, *Proc. Amer. Acad. Arts and Sci.*, 1891; also Fresenius, *Quantitative Analysis*, American edition, New York, 1886.

case the arsenic acid can be readily extracted by boiling water, and the solution filtered and evaporated to dryness. Marsh's test can be applied to the solution, and metallic arsenic will appear as a mirror on the tube or on cold porcelain.

In order to *estimate* the arsenic, the total quantity of fluid obtained as above from any organ, such as the liver, should be divided into equal parts and one or more of these parts used to get a deposit of metallic arsenic in the heated tube by the Marsh-Berzelius method. The section of coated tube is cut off and weighed, and then washed free from arsenic with nitric acid or solution of sodium hypochlorite and weighed again. The difference represents the amount of arsenic in the portion of the material used. According to Rettgers,[1] the brown deposits which are more or less transparent consist of the suboxid, As_2O, and hydrid, AsH, hence these quantitative results can never be absolutely accurate. Another method of *estimating* is by converting the arsenic into sulphid. A measured fraction of the dissolved materials acidified with hydrochloric acid is treated with a stream of pure washed hydrogen sulphid gas. The yellow precipitate is collected on a filter, thoroughly washed, and then dissolved in ammonium hydroxid. By evaporating the solution thus obtained on a water-bath, the ammonia is removed and the dried sulphid, after treatment with carbon disulphid to dissolve out any free sulphur that may be present, is weighed, and the calculation made on the basis that 100 parts of the sulphid contain 60.98 of elementary arsenic. As arsenic acid and the arsenates are precipitated very slowly by hydrogen sulphid, they will require other treatment (see p. 428).

The reaction between *silver nitrate* and arseniuretted hydrogen is the basis of another method for stating the amount of arsenic. Having started the process of hydrogen formation in the Marsh apparatus, a gas-burner should be applied to the delivery-tube to test the materials. If no stain appears on the glass tube after heating ten minutes, the apparatus may be considered arsenic-free. The end of the tube should be extended and bent down at a right angle so as to dip into a weak solution of silver nitrate. The suspected material is now poured into the flask, and the gas allowed to pass through the silver nitrate solution. The end of the process will be known by dipping the tube into a fresh sample of silver nitrate; it should not darken it. In this process the nitrate is decomposed, the free silver deposited as a black precipitate, and the arsenic trioxid will be left in solution. Any excess of silver nitrate can be removed by hydrochloric acid and filtration; then the arsenic in a fractional portion may be precipitated by hydrogen sulphid and the precipitate dried, washed with carbon disulphid, again dried, and weighed. When the arsenic trioxid is present in small quantity, the hydrogen sulphid will not make a precipitate at once, but cause a yellow color, deep in proportion to the arsenic present. A fractional portion of the tested silver solution freed from silver may be put in a Nessler cylindric glass, and a known quantity of saturated hydrogen sulphid solution added. In another cylinder the same quantity of

[1] Blyth, A. W., *Poisons, Effects and Detection.*

hydrogen sulphid solution should be put, and a weak hydrochloric acid solution of arsenic trioxid of known strength added to it from a buret until the same color is produced. The amount can then be calculated from the reading of the buret.

When the amount of organic matter present is small, as in the *urine*, the test for arsenic can be got at by a more direct method. Concentrated sulphuric acid containing one-thirtieth its volume of nitric acid is added to the urine which has been evaporated to dryness, and the residue heated until dense white fumes are given off. The charred materials are extracted with boiling water, the solution concentrated, and introduced at once into the Marsh apparatus.

Detection in Wall-paper.—When there is reason to think that the amount of arsenic is considerable, resort may be had to Reinsch's test. Five square inches of the paper cut finely are put in a dish with dilute hydrochloric acid, and heated on a water-bath for fifteen minutes. The solution is then decanted into a test-tube, a piece of polished copper-foil added, and then boiled for ten minutes. The arsenical coating on the copper can be verified by the confirmatory tests given above (p. 418).

When the amount present is so small as to give a doubtful result to Reinsch's test, it may still be revealed by the Marsh-Berzelius method. The arsenic is dissolved from the paper as stated above, and the acid solution poured into the Marsh apparatus (Fig. 41). The blackness of the arsenical mirror formed in the glass tube when compared with a series of standard arsenical mirrors will give an approximation to the quantity. Chittenden [1] has worked out a very delicate method for detection of minute quantities.

Distribution of Absorbed Arsenic.—Besides that which may be found in the contents of the stomach and intestines, a variable proportion of arsenic is absorbed, and by the blood and other fluids is distributed to different organs and tissues. This latter part has certainly had a poisonous effect, whatever may be said of that found unabsorbed. Even when none has been found in the contents or even in the structure of the stomach and the intestines, the liver, kidneys, spleen, and heart have rendered up their store to the analyst. The muscular and bony tissues and the brain are also places for the deposit of absorbed arsenic.

It not infrequently happens that the arsenic is taken in a very soluble form, and, the patient surviving for two weeks or even less, no poison can be detected in the viscera usually examined. This is due to the activity of the circulation in the soft tissues and the readiness with which the poison is eliminated. Under such circumstances several observers have discovered the poison still present in the cancellated structure of the bones and in the nails and hair. In a case reported by Wood,[2] he detected by Sanger's method arsenic in the urine ninety-three days after a single large dose had been administered, causing acute symptoms and having a sequel of paralysis. Stich [3] reported a case in

[1] Fresenius, *Quantitative Analysis*, American edition, New York, 1886.
[2] *Boston Med. and Surg. Jour.*, 1893.
[3] *Munch. med. Wochenschr.*, March 12, 1901.

which arsenic was found in the gastric contents of a woman who had been poisoned by the introduction of a large amount in the vagina. He reported another case of arsenic in the gastric contents and feces of a woman, and some was obtained from the organs of her three months' fetus.

The case of Jennie Cramer is very important in medicolegal annals, showing that in order to make an estimate of the amount present in the body, it is necessary to analyze not only the internal organs, but specimens of the muscles, bones, nails, and hair must be included. The post-mortem revealed no evidence of inflammation of the stomach. Still the prosecution held that though the body was found in the water, as arsenic was discovered in the brain and other tissues, death probably occurred not from drowning, but from the sudden cerebral impression produced by the poison having been taken in a very diffusible state. A few days after death the analysis was made and the following instructive illustration of distribution was obtained, as stated by Chittenden:[1]

The calculation is in grains of arsenic trioxid, As_2O_3: Stomach and esophagus, 0.158; liver, 0.218; intestines, 0.314; kidneys, 0.029; heart, 0.112; lungs and spleen, 0.1528; brain, 0.765; trachea, larynx, and tongue, 0.081; diaphragm, 0.010. The internal organs yielded a total of 1.1694 grains. From different parts of the body the muscular and bony structures, with a total weight of 24 pounds 14½ ounces, were subjected to the same analysis, with the result of finding 0.852 grain of arsenic. With these data it was calculated that the entire body, including the viscera, contained a total of 3.1192 grains. While it strengthens a case to prove that the corpse contains a fatal quantity, it is not essential for conviction, as shown in the trial of Mrs. Maybrick, when only one-third of a grain was found in the viscera analyzed.

Failure to Detect.—Instances are known of undoubted poisoning in which no trace of the arsenic has been found in the parts usually examined. The eliminating organs had time before death to expel the unnatural substance.

Normal Arsenic.—Gautier[2] found arsenic in the tissues of the thyroid and thymus glands, the skin, and the brain, chiefly in the form of nuclein iodid. Later[3] he reported it constantly present in the fresh thyroid of man. A trace was found in the hair of a man and also of a woman, neither of whom had ever taken arsenic. Some was detected in the thymus of a lamb. Traces were discovered in the mammæ of a cow and in two quarts of her milk. Fresh bone furnished a trace. The brains of two still-born children showed its presence, but it was absent in a third. He failed to find arsenic in the presumably healthy tissue of liver, kidney, spleen, muscles, testicles, pituitary gland, mucous membrane, cellular tissue, lymphatics, salivary glands, suprarenal capsules, bone-marrow, uterus, ovaries, blood, urine, and feces. Upon examining

[1] *Amer. Chem. Jour.*, vol. v., No. 1.
[2] *Bull. de l'Acad. de Méd.*, December 5, 1889.
[3] *Ibid.*, Feb. 5, 1900; also *Comptes Rendus Academie des Sciences*, vols. cxxix., cxxx., cxxxi.; vol. cxxxiv., p. 1434; vol. cxxxv., pp. 809 and 833; see also article by Bertrand, *Annales de l'Institut Pasteur*, July, 1902.

various foods he found it absent from bread, fish, eggs, and meats, excepting the tissues named above—viz., milk, thymus, thyroid, skin, and brain. It was present in the following vegetables: cereals, turnips, cabbages, and potatoes. In case arsenic should be found in the tissues named above as normally free from it, the inference would be that arsenic had been taken as a medicine or criminally.

Dearden[1] reports that he found measurable quantities of arsenic in one gram of the hair of several persons having arsenical neuritis, and a trace in the same amount of hair from healthy subjects.

Hödlmoser[2] reaches a conclusion opposed to that of Gautier. His research showed that arsenic is not a normal constituent of the human body. He did not find a trace of arsenic in the liver and pancreas in 18 cases examined by Gautier's method, nor in 15 other cases in which the same viscera were examined by another process esteemed more delicate. In repeating the experiments of Gautier referred to on different tissues, he always obtained negative results.

Arsenic Pentoxid (Chemical Formula, As_2O_5; Synonym, Arsenic Acid).—Arsenic pentoxid, As_2O_5, commonly occurs in white, vitreous, deliquescent masses, but may be obtained as rhombic crystals. The pentoxid deliquesces in the air and changes to the true acid, $As_2O_5 + 3H_2O = 2H_3AsO_4$. This is a colorless, acid, syrupy liquid with a metallic taste. Like arsenic trioxid, it is an irritant poison. The free acid is not used in medicine, but some of its salts are. It is much used in the manufacture of dyes, although recently other oxidizers have supplanted it to a considerable degree. It responds to the same tests as arsenic trioxid, but has a peculiar reaction with silver nitrate, forming a brick-red precipitate (No. 4, Plate 6). It will respond to Marsh's test, but it is precipitated very slowly by hydrogen sulphid. If there is reason to think that either arsenic acid or any arsenate is present in the tested fluid, it should be reduced to arsenous acid by a current of sulphur dioxid and the latter removed by passing carbon dioxid before the hydrogen sulphid is added to it. A solution of sodium arsenate, four pounds to the gallon, is commonly injected by undertakers through the nostrils into the stomach and into the thoracic cavity in order to arrest decay in warm weather. Sometimes cloths are wet with it and wrapped about the corpse to accomplish the same end.

Presence of Arsenic in Various Substances.—*Medicinal Preparations.*—The acidum arsenosum of the pharmacopeia (white arsenic, As_2O_3) is present to the amount of 1 per cent. in each of the following preparations: *Liquor acidi arsenosi; liquor potassii arsenitis* (Fowler's solution); *liquor sodii arsenitis* (Harle's solution). Donovan's solution, or *liquor arseni et hydrargyri iodidi*, contains 1 per cent. each of arsenous iodid, AsI_3, and mercuric iodid, HgI_2, while Pearson's, or *liquor sodii arsenatis*, contains 1 per cent. of sodium arsenate. Sodium cacodylate, $As(CH_3)_2O_2Na$, while generally well borne in large doses, may undergo changes when given by the stomach or the rectum and have toxic effects.[3]

[1] *Lancet*, 1901. [2] *Zeitschr. f. physiolog. Chem.*, October, 1901.
[3] *Brit. Med. Jour.*, 1900, vol. ii., p. 823.

It contains arsenic in an organic form and very high proportion. An arsenical paste has been applied to tumors by cancer quacks so unskilfully as to produce systemic poisoning by absorption. The manufacturers of gelatin-coated and sugar-coated pills sell large quantities of tonic pills containing arsenic as a constituent. It is often a contaminant of commercial glycerin and of subnitrate of bismuth. In 1 out of 8 samples of bismuth subnitrate examined, Coad[1] found arsenic; it was present to the amount of 0.33 per cent. of the element. In this preparation it probably exists as bismuth arsenate, a form not readily absorbed because of its insolubility.[2]

In Preservatives and Cleaners.—In order to keep wheat for planting it has been treated with an arsenical solution which does not alter its appearance or taste. Samples so treated have caused accidental poisoning. A mixture of white arsenic, tar, and soft soap is sometimes used as a "sheep dip" to destroy the parasites in wool. The sheep-washers have experienced poisonous effects from handling it and from drinking water from a vessel that once contained it. Taxidermists make use of an arsenical soap and an arsenical powder to preserve skins. The workmen have suffered, and it is stated that poisonous symptoms can be traced to the arsenic emanating from stuffed specimens kept in sleeping-rooms. Wholesale poisoning has followed the introduction, into a boiler, of a "cleaner" made from arsenic and sodium bicarbonate. A similar solution has been used as a "soft injection" or preservative of bodies for dissection. Dissectors handling the bodies are likely to have a local irritation about the finger-nails.

In Anilin Dyes.—Arsenic acid or arsenic pentoxid is frequently used as an oxidizer by the color-men in the preparation of anilin-red and other pigments. It is not a necessary ingredient of the pigment, and at this time is not so often found in it as formerly. The expense of washing out this residuum still sometimes deters the manufacturers, and the dye may come into the market with enough arsenic in it to give irritant properties to stockings, gloves, and cretonne bed-trimmings reddened with it. This impure anilin-red has been used to color strawberry and raspberry syrups.

In the Air.—In the chemical laboratories eight deaths[3] have occurred from the accidental inhalation of the vapor of arsenuretted hydrogen escaped from the Marsh apparatus. This is the most deadly of all the inorganic compounds of arsenic. It has destroyed life in quantities so small as not to impart a garlicky odor to the air. Traces of this poison have been found in common illuminating gas. By some writers this is the form supposed to be taken by the arsenic emanations of wall-paper, though other authorities suppose the emanation to be an organic compound, such as cacodyl oxid. In the extraction of silver from certain ores, the cleaning of iron for tinning and of brass for bronzing, acids are used which liberate hydrogen. This nascent hydrogen unites with

[1] *Amer. Chem. Jour.*, New York, 1875. [2] *Ibid.* 1881–82, vol. iii.

[3] Taylor, A. S., *On Poisons*, 1875; Witthaus and Becker, *Medical Jurisprudence, Forensic Medicine, and Toxicology*, 1896.

arsenical impurities in the metal, if any is present, and thus poisons the air breathed by the workmen.

In Beer.—In the health reports of France of 1878 it was said that the glucose used in brewing sometimes contained arsenic. It is left in the glucose by the sulphuric acid employed in the conversion from starch, when the acid is obtained from arsenical iron-pyrites. The *Lancet* (December 1, 1900) analyzes reports from many sources of extensive poisoning in Liverpool, Manchester, and the Midland counties of England attributed to arsenic in beer, and extending through six months. The most frequent characteristic symptoms were catarrh, puffiness of the eyelids, irregular pigmentation of the skin, herpes and other eruptions, local numbness, tingling, and pain, with final paralysis. Arsenic was detected in the urine and in the hair. The amount of arsenic found in different samples of beer varied from $\frac{1}{100}$ to $\frac{8}{10}$ of a grain to the gallon.

In the Household.—Many cases of poisoning, accidental or otherwise, have been traced to things in common domestic use. "Fly-papers" for killing flies are sheets of paper saturated with sweet solutions or pastes of arsenic. Single sheets have been examined which contained ten grains of arsenic trioxid available for the poisoner. "Fly-powders" have been made by pulverizing the mineral arsenid of cobalt. "Buffalo carpet-moth annihilator," intended to be dusted over the carpet, is a powder containing arsenic. White arsenic is often mixed with a dough of flour or corn-meal and distributed in cellars and pantries to kill mice. The most extensively used domestic vermin-killer is "Rough on Rats," a mixture of white arsenic with charcoal. It is a gray powder put up in packages and procurable in every drug-store without restraint or legal registration. In this country a large number of suicides and some homicides have been caused by this agent. Among the means for wilful death it appears to be our national favorite. It is cheap, knowledge of its deadly properties is common, and there is every facility for purchasing it under the excuse of killing vermin. It is much to be desired that our state and municipal legislation relative to the sale of arsenical commodities should be shaped after the pattern of other civilized countries. Some provision should be made in the laws of every state which would require apothecaries to keep an arsenic book for recording sales of this poison, and which would forbid the sale of arsenic in any shape for the purpose of destroying vermin or for the embalming of dead bodies.

To overcome the gypsy moth the white precipitate of lead arsenate, Pb_32AsO_4, is applied to the foliage of plants. "Arsenical balls" are given by grooms to improve the coats of horses. Some kinds of white enamel-ware and some glass contain arsenic. It has been found in the silk lining of coat sleeves, the glazed leather lining of hats, the brown paper lining of carpets, and the black cambric lining of furniture. It is sometimes present on the glazed paper and cardboard used for boxes, playing-cards, note-paper, and fancy wrappers for candy lozenges. At one time arsenical pigments were extensively used for coloring wall-hangings, lambrequins, cretonnes, chintzes, tarlatans, and artificial flowers and leaves. It has been alleged that numerous cases of slow poisoning have

been traced to the arsenic from these sources pervading the atmosphere of dwellings. In making a diagnosis reliance has been placed upon the discovery of traces of arsenic in the urine of patients. It has been found there with surprising frequency.[1] Harding[2] records a group of cases of chronic poisoning among the nurses at an asylum, traced to the green baize curtains, which were found to contain a large amount of arsenic. Kirschgässer[3] has reported 26 cases of apparent poisoning from this source, in most of which, by collecting the urine for two days and evaporating it, he found arsenic present.

The possibility of poisoning under these conditions has been disputed by Pellew and by Chandler.[4] The question is clouded by the liability to a fallacy from the presence of traces of arsenic in such common materials as the illuminating-gas and even the filter-paper used by the analyst. It has been found in the urine of persons apparently healthy. Kossa[5] has noticed traces of arsenic, copper, and mercury in the urine of numerous healthy individuals examined. He is inclined to think that these substances are not totally eliminated by the organism, and that in time the accumulated amounts might have an important bearing in certain medicolegal cases.

Hills[6] analyzed the urine for arsenic in 180 cases, many of which were without symptoms of arsenical poisoning. He found arsenic in 75 per cent., showing coincidence, rather than causation, of disease. The process of elimination by the kidneys was extremely slow. The daily amount averaged less than 0.01 mg. a liter of urine. In some cases as many as eighty days elapsed before arsenic disappeared from the urine. The use of Paris green as an insecticide in gardens and the presence of arsenic in coal-gas may be sources from which minute traces are derived in unsuspected ways.

It would appear to be necessary to conduct blank experiments in these analyses before drawing conclusions. Certainly the evidence of a minute trace of arsenic would be much more valid if positive assurance could be given that all the materials were absolutely free from arsenic. Making all allowances for sources of error, it is clear that cases do occur of domestic poisoning presenting characteristic symptoms and yielding unmistakable proof of arsenic in the urine. The theory of the gaseous form of the poison has received much support from the experiments of Gosio and Sanger. Gosio's experiments at the Public Health Laboratories at Rome proved that a volatile arsenical compound was produced by certain common moulds growing in contact with arsenicated substances such as might be used in wall-papers. Since the researches of Hamburg and Bischoff some have supposed this to be arsenuretted hydrogen developed by the nascent hydrogen set free by the fermenting mould.

[1] A full account of some cases is given in Report of the Massachusetts State Board of Health for 1885; others were reported in *Boston Med. and Surg. Jour.*, 1887 and 1889, and in the *Edinb. Med. Jour.*, 1887.

[2] *Lancet*, 1892.

[3] *Vierteljahresschr. f. ger. Med.*, 1868, new series, vol. ix.

[4] Hamilton, A. M., *Legal Medicine*, New York, 1894.

[5] *Wien. klin. Rundschau*, No. 33, from the *Ungar. Arch. f. Med.*, No. 8.

[6] *Boston Med. and Sur. Jour.*, 1894.

Professor Sanger[1] has recently repeated Gosio's experiments and substantiated his results. He found that, with moisture, a temperature between 60° and 95° F. (15° and 35° C.), a supply of oxygen, and a small amount of arsenic, there was a development of a volatile product containing arsenic. It was not arsenuretted hydrogen, but, in Sanger's view, an organic derivative of arsenic pentoxid. The conflicting results obtained by Chandler are accounted for either by his using too large an amount of arsenic or by the absence of the particular moulds required. These were the *Penicillum brevicaule*, *Muco mucedo*, *Aspergillum virens*, and *Aspergillum glaucum*. Some investigators who wrongly assumed that the product would be arsenuretted hydrogen and limited their inquiry to this agent, have had negative results. In the present state of knowledge no wall-paper containing as much as 0.1 grain of arsenic in a square yard can be considered as harmless.

In Common Pigments.—Both accidental and intentional poisoning occur from the use of the pigments described below:

"*Scheele's green*" (copper arsenite, $CuHAsO_3$) contains 52.8 per cent. of arsenous acid. A bright-green paint is made by mixing this with the basis of oil-paints and of water-colors. Although the public is warned as to the deadly character of this pigment, it is still much used for giving color to wax tapers, toys, book-covers, artificial flowers, oil-cloth, calicoes, cretonnes, and tarlatan. An equivalent green color and one much less injurious can be made by mixing Prussian-blue and chrome yellow.

"*Paris green*" (copper aceto-arsenite, Schweinfurth-green) is a color made by mixing the acetate of copper with the arsenite. It contains over 50 per cent. of arsenic. There is an enormous consumption of this compound as an application to the potato plant to rid it of the Colorado beetle. Used on the tops, this does not affect the edible tuber under ground. The same practice upon the tobacco-plant is far from innocent, as the leaves here are the parts to be used. It is often taken by suicides, but its color usually prevents its criminal use, though occasionally accidental death has been caused by it.

Realgar (As_2S_2); *Orpiment* (As_2S_3); "*King's Yellow.*"—The first of these arsenic sulphids occurs in ruby-red crystals containing 70 per cent. of arsenic; the second, containing 61 per cent., is a yellow powder which, by mistake, has sometimes been substituted for the harmless vegetable pigment, turmeric. The action is similar to that of white arsenic.

Arsenic in the Soil.—Some soils naturally contain arsenic in an insoluble iron compound. This compound is incapable of yielding arsenic to a corpse buried in it. It has been stated above that arsenic is frequently present in commercial sulphuric acid. This acid is used in the manufacture of superphosphate and other artificial manures, which contribute it to the soil. From the soil it may be taken by our food-plants, such as potatoes and turnips. In cities it is the general custom for undertakers to embalm corpses by pumping a solution of sodium

[1] *Proc. Amer. Acad. Arts and Sci.*, vol. xxix.

arsenate through the nostrils into the stomach, trusting to the high diffusibility of that salt to carry it throughout the body. The experiments of Witthaus[1] show that in twelve days the arsenic may permeate the entire body, reaching the brain. It is possible that this same compound would eventually pervade the soil of the cemetery contiguous to a buried corpse.

ANTIMONY.

(Chemical Symbol, Sb; Synonym, *Stibium.*)

Antimony is a brilliant gray-white solid with a crystalline, metallic fracture, tasteless and odorless. When heated it volatilizes; at a higher temperature it burns to antimony trioxid (Sb_2O_3). It is used as an alloy in type-metal, Brittania-metal, brass, and bell-metal. Though the metal may not be poisonous, its salts are.

While poisoning from antimony was quite common in the middle ages, in our times it is comparatively rare. Cases have been reported (Lohmeier) from inhalation, probably of the trioxid, in certain industries. Lozenges containing the same preparation were the cause of poisoning in another case.[2] In modern toxicology but two forms figure to any extent, the trichlorid and tartar emetic.

Sulphurated antimony, a mixture of Sb_2S_3 and Sb_2O_3, is employed in vulcanizing rubber. The India-rubber connections of the Marsh apparatus might thus contribute a trace of antimony unless care be taken to avoid the use of fittings made with this preparation.

Antimony Trichlorid ($SbCl_3$; *Butter of Antimony*) occurs as a strong solution of the chlorid in hydrochloric acid, and is employed in the arts as a *bronzing* liquid, in farriery, and in medicine rarely as an external application.

The records of 8 cases of poisoning have been studied by Witthaus,[3] who found that in the 4 fatal ones the dose was 2 ounces, while 2 that recovered took 1 ounce each. A woman of forty years[4] died in less than two hours; in her stomach were found 8 grains of antimony and 0.1 grain of arsenic.

Tartar Emetic (Chemical Formula, $KSbOC_4H_4O_6$; Synonyms, *Tartarated Antimony; Stibiated Tartar; Antimony and Potassium Tartrate*).—This is a white crystalline powder with an acrid, disagreeable, metallic taste. It is made by the action of a boiling solution of cream of tartar upon antimony trioxid. It has been dispensed by mistake for cream of tartar and for tartaric acid. It is soluble in cold water, more readily in hot water, but insoluble in alcohol. Wine is used as a vehicle in *vinum antimonii*, the water of the wine acting as a solvent and the alcohol checking the formation of the moulds, to which a simple aqueous solution is liable. It is present in *syrupus scillæ compositus* ("Hive Syrup") and in *unguentum antimonii*. According to Witthaus,[5] who

[1] *Bulletin of Loomis Laboratory*, 1890. [2] *Lancet*, 1879.
[3] Witthaus and Becker, *Medical Jurisprudence, Forensic Medicine, and Toxicology*, New York, 1896.
[4] Witthaus and Becker, *ibid.*, 1883.
[5] Witthaus and Becker, *ibid.*, New York, 1896.

Fatal Dose.—The smallest dose that has proved fatal to a child is $\frac{3}{4}$ grain (48.5 mg.).[1] A healthy woman aged twenty-five years took the maximum medicinal dose, $1\frac{1}{2}$ grains (97.2 mg.), without effect, but a similar dose twenty-four hours later excited violent purging and vomiting, with death in thirty-six hours.[2] Such cases cannot be considered as fixing the danger limit. Ten grains[3] at one time would be a dangerous dose, but the same amount in broken doses would be still more so.

Recovery has followed a dose of 170 grains.[4] As a rule, prompt emesis follows the administration of a large dose, and the effects are mainly local and not serious. If the poison be retained and absorbed, the vomiting center is indirectly involved, and purging, with extreme depression, becomes the prominent symptom. At one time it was considered good practice in acute inflammatory diseases to give doses of a grain at intervals to establish "tolerance." By the second day some patients would tolerate the drug without vomiting and purging, and "heroic" doses of five grains each could be given without inducing these effects. As much as sixty grains daily have been given in this way without disturbance of the stomach. The effects in such cases are mainly those of depression of the heart's action and of the nervous system.

Fatal Period.—A fatal result has occurred in an adult in seven hours. In the exceptional case reported by Taylor death occurred in a child in three-quarters of an hour. The fatal event may be delayed for several days, the average duration of life being twenty-four hours.

Treatment.—As a rule, the free vomiting induced by the tartar emetic is sufficient to evacuate the stomach. In the rare cases where it does not occur other emetics should be given, such as sulphate of zinc or mustard and water; or the stomach may be washed out with a mixture of hot water with the antidote, tannic acid; or a decoction of green tea or of some vegetable astringent—all these forming the insoluble tannate of antimony. When the stomach has been emptied, morphin should be given hypodermically to relieve pain, and the irritable stomach and bowels treated with suitable remedies. The depression of the heart must be counteracted with stimulants, aided by dry heat or mustard to the epigastrium and the extremities.

If antimony chlorid has been taken, the corrosive action on the stomach would cause a condition which would be aggravated by the mechanical irritation of the stomach-tube.[5]

Postmortem Appearances.—One of the victims of Dr. Pritchard, Mrs. Taylor, died from acute tartar-emetic poisoning, and the autopsy revealed nothing, although the poison was found in the viscera, urine, blood, and intestinal contents. Such a result is quite exceptional, most cases showing redness, swelling, ecchymotic patches, and perhaps ulceration in the gastro-intestinal mucous membrane. Sometimes the changes in the gullet and pharynx are profound, as in a case described by

[1] Taylor, *Guy's Hosp. Reports*, 1857.
[2] *Bull. de Thérap.*, vol. ii.
[3] Taylor *On Poisons*, etc., 3d ed., 1875.
[4] Carpenter, *New York Med. Record*, 1888.
[5] Compare p. 310 in section on General Principles of Toxicology.

Blyth,[1] in which there was destructive ulceration of the membrane of the epiglottis and of the adjacent parts, exposing the muscular fibers of the pharynx. In a case recorded by Cook,[2] of poisoning from the corrosive antimony chlorid, after vomiting without blood the patient went into collapse and died in two hours. The gastric membrane was almost black from congestion.

In cases of chronic poisoning it is usual to find inflammation of the kidneys and liver.

Tests.—*1. Hydrogen Sulphid.*—A stream of this gas will precipitate orange-red antimony trisulphid (Sb_2S_3) when passed through any antimonial aqueous solution acidified with hydrochloric acid (No. 5, Plate 6). This orange precipitate is insoluble in ammonium hydroxid, but dissolves in the fixed alkalis, in ammonium sulphid, and in strong hydrochloric acid, especially when heated. A very characteristic reaction is obtained when this hydrochloric acid solution (after boiling to expel all trace of hydrogen sulphid) is diluted with excess of water. A white precipitate of antimony oxychlorid falls, which is soluble in tartaric acid.

Fallacies.—While this test is quite certain in simple solution, it may give a doubtful result in the presence of colored organic materials.[3] These should be entirely destroyed to give a satisfactory verdict.

Delicacy.—A definite reaction can be obtained with 1 : 10,000 of a grain of antimony trioxid in five grains of solution.[4]

2. Reinsch's Test.—The method of performing this test has been described under the heading of Arsenic. If any precipitate forms when the suspected solution is acidified with hydrochloric acid, more acid must be added until the oxychlorid is redissolved. On boiling in it a strip of bright, pure copper-foil a film of metallic antimony will appear. If the amount is small, the film is violet. A larger quantity will give a surface like tarnished zinc, and if abundant, a black amorphous layer.

Fallacies.—Arsenic, mercury, and some other metals make similar deposits. To distinguish the nature of the metallic films the copper strip must be washed, dried, coiled, and heated in a glass tube open at both ends. Under this treatment antimony yields a white sublimate of antimony trioxid, which is usually amorphous, although sometimes showing crystals; arsenic gives a sublimate of octahedral crystals; mercury a sublimate of shining metallic globules; and other metals, as a rule, produce no sublimate. The antimony trioxid may be dissolved in weak tartaric acid and an orange-red precipitate be obtained by passing hydrogen sulphid after acidification with hydrochloric acid. Again, the film of antimony on copper may be identified by boiling it in a weak solution of potassium hydroxid, removing the strip at intervals to expose it to the air. If the solution of antimony thus made is acidified with hydrochloric acid, it will yield an orange-red precipitate with hydrogen sulphid.

[1] *Poisons: Their Effects and Detection*, p. 553. [2] *Lancet*, 1883.
[3] Reese, "Wharton Trial," *Amer. Jour. Med. Sci.*, 1872.
[4] Wormley, *Microchemistry of Poisons*, 1886.

Delicacy.—A distinct violet-colored deposit on the copper can be obtained from one grain of a solution containing $\frac{1}{20000}$ grain of tartar emetic,[1] or $\frac{1}{50000}$ of a grain of antimony trioxid.

3. Marsh's Test.—Under the section on Arsenic details have been given for performing this test. If antimony be present, the gaseous terhydrid will be formed, which has not the onion-like odor of arsenic terhydrid. Its flame produces a black spot on cold porcelain, while a metallic mirror forms in the delivery tube if that be heated by Berzelius' method. These may be mistaken for the similar deposits made by arsenic. When treated with solution of chlorinated lime or chlorinated soda, the antimony deposit is insoluble, while arsenic dissolves. Yellow ammonium sulphid dissolves both, but on evaporation the solution of antimony sulphid leaves an orange-red spot soluble in strong hydrochloric acid, but insoluble in ammonia. The corresponding arsenic sulphid is yellow, insoluble in hydrochloric acid, but soluble in ammonia.

If the gas, instead of being burned or decomposed by heat as above, is passed into solution of silver nitrate, there is a black deposit of silver antimonid, Ag_3Sb. If arsenic is also present, it remains in solution and, by filtration, we can separate the two. The filtrate can be tested for arsenic. The antimony in the precipitate may be separated from the silver by dissolving in boiling weak hydrochloric acid. When filtered again and treated with hydrogen sulphid, the filtrate gives orange-red antimony sulphid.

Delicacy.[2]—With a small apparatus spots on porcelain are obtained from 50 grains of a fluid containing $\frac{1}{200}$ grain of antimony trioxid, while a good deposit in the heated tube is yielded by the same amount of fluid containing $\frac{1}{1000}$ grain of antimony trioxid.

The *silver nitrate test* gives a still more delicate reaction, and can be obtained with only a few drops of the test solution, a satisfactory deposit of silver antimonid forming when there is present only $\frac{1}{8000}$ grain of tartar emetic, equal to $\frac{1}{20000}$ grain of antimony trioxid.

4. *Zinc Test.*—The suspected liquid is put into a platinum dish and acidified with hydrochloric acid. On immersing a slip of pure zinc, the antimony, but not arsenic, is at once deposited on the platinum as a black stain, which can be removed later by nitric acid or by simple heat. The true nature of this stain is revealed by wetting it with nitric acid, drying at a gentle heat, and touching with a drop of dilute ammonium sulphid, when an orange-red color will be produced, due to the formation of antimony trisulphid.

Delicacy.—This test is stated by Fresenius[3] to be very delicate. In two minutes a brown stain will appear when the solution holds but $\frac{1}{10000}$ grain of antimony, a definite reaction showing in a quarter of an hour when the amount is only $\frac{1}{20000}$.

5. *Tin Test.*—If an antimony solution is acidulated with one-tenth

[1] Wormley, *Microchemistry of Poisons*, 1885.
[2] Wormley, *ibid.*, 1885.
[3] Fresenius, *Quantitative Analysis*, American edition, New York, 1886.

part hydrochloric acid and a slip of pure tin-foil is immersed in it, the foil turns black from a deposit of metallic antimony.

Detection.—*In Vomited Matters and Urine.*—Owing to the prompt action of tartar emetic, the stomach and bowels are usually quickly evacuated of the poison. A large part of that which is absorbed into the general circulation is rapidly eliminated by the kidneys, and hence the proportion stored in the viscera is relatively small. In cases of suspected chronic poisoning the vomited matters, the liquid feces, the urine, or medicinal mixtures should be subjected to analysis. For this purpose the material is acidified with hydrochloric acid, and the zinc test or Reinsch's test applied. These respond even in the presence of organic matter. To another portion of the material, acidified with hydrochloric acid, tartaric acid is added; it is heated on a water-bath for half an hour, strained, filtered, and the filtrate treated with a stream of hydrogen sulphid for several hours. The precipitate, which may contain the sulphids of certain other metals and free sulphur, should be treated with strong hydrochloric acid and boiled as long as hydrogen sulphid fumes escape. The filtered solution may be tested with Reinsch's test, the zinc test, or Marsh's test, collecting the antimony in silver nitrate solution. In testing the urine, the total quantity for several days should be evaporated to a small bulk before being operated on.

Separation from the Tissues.—From the solid viscera most of the antimony can be extracted by mincing a portion and boiling it for an hour in water, 5 parts, acidified with hydrochloric acid 1 part. The strained and filtered solution may be tested by Reinsch's or the zinc test.

Quantitative Determination.—If it is desired to calculate the total amount of antimony, it is best to use the process for destruction of organic matter by hydrochloric acid and potassium chlorate given under the section on Arsenic.[1] This being done, the mixed precipitate obtained by passing hydrogen sulphid through the acidified fluid is washed, treated with strong nitric acid, and evaporated to dryness. A small quantity of a strong solution of potassium hydroxid is added to the residue, it is filtered, evaporated to dryness, and fused. The potassium antimonate in this fluid is boiled with solution of tartaric acid, acidulated with hydrochloric acid, filtered, and saturated with hydrogen sulphid gas. The orange-red antimonic sulphid (Sb_2S_5) thus obtained is washed on a Gooch filter, dried in a water-oven, and the free sulphur and residual moisture which are always present expelled by heating in an atmosphere of dry carbon dioxid. Of this residue, which has been converted to the black sulphid (Sb_2S_3), 100 parts represent 71.77 of antimony.

When the presence of other poisonous metals is suspected, the precipitate made with hydrogen sulphid is treated thoroughly with yellow ammonium sulphid and the solution filtered. The arsenic or antimony is present in this filtrate, while mercury, lead, and copper remain upon the filter to be examined by appropriate methods. The filtrate is evap-

[1] Consult also p. 327 in section on General Principles of Toxicology.

orated, and the residue treated with nitric acid and potassium hydroxid to convert the metals into potassium arsenate and antimonate. If the presence of both metals is suspected, this mixture is put into the sulphuric acid and zinc Marsh apparatus and the gas passed through silver nitrate as long as a black precipitate falls. The arsenic will be in the solution, and is separated by filtration. The black antimonid of silver is collected on a filter, washed, boiled with tartaric acid, acidulated with hydrochloric acid, filtered, and the filtrate precipitated with hydrogen sulphid. Of the dried precipitate of orange sulphid, 100 parts represent 196.47 parts of pure crystallized tartar emetic.

TIN.

(Chemical Symbol, Sn.; Synonym, *Stannum.*)

Though poisoning from tin salts is rarely reported, there is sufficient evidence to prove that it does occur.

Putty powder, a higher oxid of tin, was the cause of death in the case of a chemist,[1] who, by mistake, used it for months in his pepper-box. The solder used for fruit-cans contains tin. This, as well as the tin surface, may be dissolved by the action of acid juices of fruits or the fatty acids of meat and cause toxic symptoms. In the case of canned meats, the danger from this source is slight. According to Rössing,[2] it is unworthy attention, as the compound formed is insoluble in the digestive juices. He thought that the traces found in canned meat and fish existed as oxid. According to Wirthle,[3] it is sometimes a basic stannous chlorid and sometimes a sulphid. He found that the corrosion of the tin can increase slightly after the second year, but the amount is never anything but slight, even after four years.

Symptoms.—Tin salts act as gastro-intestinal irritants, causing sometimes a metallic taste, usually nausea, vomiting, abdominal pain, diarrhea, cyanosis, and collapse. Severe symptoms like these were seen in 4 cases described by Luff and Metcalfe,[4] due to eating tinned cherries, the strongly acid juice in the can showing 3.2 grains of malate of tin to the fluidounce.

In an investigation by Ungar and Bodlander [5] upon the lower animals it was shown that even the non-irritating salts given subcutaneously caused toxic symptoms like those of other metals which undermine the health, sometimes even causing death. They found tin in the tissues and the urine.

Treatment.—Emetics and the stomach-pump should be used first, followed by bland demulcent drinks, stimulants, and anodynes.

Tests.—*Hydrogen sulphid* yields with stannous solutions brown stannous sulphid; with stannic solutions, yellow stannic sulphid (No. 6, Plate 6).

Mercuric chlorid is reduced by stannous chlorid to a gray deposit of metallic mercury.

Solutions of fixed alkalis give with stannous salts a white precipitate

[1] *Med. Press and Circ.*, 1894.
[2] *Zeitschr. f. allgem. Chem.*, 1900.
[3] *Chem. Zeitung*, 1900, vol. xxiv.
[4] *Brit. Med. Jour.*, 1890.
[5] *Chem. Centralbl.*, 1887.

of hydroxid which is dissolved by excess, and on boiling is reprecipitated as black oxid. With stannic salts the white precipitate when dissolved is not reprecipitated by boiling.

Detection.—To extract the metal from the tissues and organic fluids they should be boiled for some time in water acidulated with hydrochloric acid, filtered, and the above tests applied to the filtrate.

Illustrative Case.—A. Rolles[1] reported motor and sensory disturbances in the lower extremities of a young woman whose lower extremities were at the same period stained yellow from wearing yellow silk stockings. More marked nervous symptoms like ataxia were noted a few weeks later simultaneous with wearing the stockings. The urine was albuminous, and gave the tin reaction for two months after the stockings had been laid aside. The stockings were heavily impregnated with tin chlorid to give them "body," and the absorbed tin had produced marked anemia. With the exception of hysteric symptoms, the patient recovered in a few months.

CHROMIUM.

(Chemical Symbol, Cr.)

Toxic effects have resulted from potassium chromate, from chromic acid, and from lead chromate. As the poisonous properties of lead chromate, "chrome yellow," are mainly due to the lead contained in it, they are properly considered under the compounds of lead.

Potassium bichromate occurs in orange-red crystals used by dyers and furniture-stainers. Operatives in chemical works find that in the shape of fine aërial particles it irritates the respiratory passages, sets up ozena, and causes eruptions and excoriations leading to chronic ulcers.

Chromic acid (CrO_3), chromic anhydrid, occurs in crimson prismatic needles, deliquescent, freely soluble, and in strong solution acts on organic matter with energy. Its only use in medicine is external, as a deep caustic to the tonsils and to papillary growths. When applied to fungous growths in the mouth a portion may be swallowed, as in the case reported by Tisne,[2] in which a dentist used it too freely. It caused an acrid taste and burning in the throat, pain in the nucha, with persistent vertigo, abundant vomiting of a ropy green fluid, and great prostration, with eventual recovery. Chromium was found in the urine. Even its external use is attended with danger. This was shown in the case reported by White.[3] One application of about 50 grains in half an ounce of water was made to the external genitals after removal of papillary vegetations. In twenty-seven hours the woman died in a state of collapse. Congestion of the kidneys and liver was found, and both organs contained chromium.

Symptoms.—When swallowed, the compounds of chromium act as gastro-intestinal irritants, with additional effects upon the central nervous system. They cause a disagreeable taste, vomiting, pain, diarrhea, collapse, unconsciousness, dilated pupils, very slow respirations, and muscular cramps.

[1] *Wien. klin. Rundschau*, March 10, 1901. [2] *Jour. de Méd.*, Paris, 1887.
[3] *University Med. Mag.*, 1889.

Fatal Dose.—Maschka has described a case of suicide in fourteen hours from about 3 drams of potassium bichromate, while Macniven[1] has reported a case of recovery from about 3 drams, and Philipson[2] a case of recovery from 273 grains.

Fatal Period.—Death from an ounce has occurred in forty minutes.

Treatment.—Chalk or magnesia should be given to neutralize the acid. Milk may be administered or used to wash out the stomach with the pump or tube. Anodynes are indicated for the pain, cerebral and respiratory stimulants for the depression of the nervous system.

Postmortem Appearances.—In his research Pander[3] found that chromic acid preparations were absorbed with great rapidity both by stomach and skin, and its elimination was mainly by the kidneys, but to some extent by the liver and bowels. In acute cases death was caused by respiratory arrest or central nervous disturbance. In the gastro-intestinal tract were found inflammation, ecchymoses, and swollen follicles. An early morbid change was parenchymatous nephritis; the spleen was shrunken and the blood altered.

Tests.—Soluble chromates yield with silver nitrate a red precipitate; with lead nitrate, a yellow precipitate; with boiling dilute sulphuric acid and alcohol, a green color.

Detection.—Having treated organic matters with hydrochloric acid and potassium chlorate, the liquid turns green from chromic chlorid. Ammonium hydroxid added to the filtered liquid in slight excess will yield hydrated chromic oxid as a precipitate, which, after washing and drying, can be converted into potassium chromate by fusing with potassium nitrate and carbonate. After dissolving the fused mass (which will be more or less yellow in color if chromium is present) in water and making slightly acid with acetic acid, the chromate can be detected by the tests given above.

IRON.

(Chemical Symbol, Fe; Synonym, *Ferrum*.)

Iron is present in the body, notably in the blood-coloring matter, also in food, and is a frequent constituent in tonic medicines. Sufficient evidence exists that at least two of its salts, ferrous sulphate and ferric chlorid, have toxic properties.

Ferrous sulphate ($FeSO_4,7H_2O$), popularly known as "copperas" and "green vitriol," occurs in coarse green crystals often covered with a brown, rusty coat. In an unsuccessful attempt at suicide[4] a woman took two ounces on an empty stomach. Functional nervous disturbances soon appeared, but subsided in a day or two, while diarrhea and abdominal pain marked the course of a subacute gastro-enteritis which lasted more than a month.

Ferric chlorid (Fe_2Cl_6) occurs most commonly in the widely used preparation *tinctura ferri chloridi* or "tincture of iron," a brown acid liquid, frequently mistaken for harmless liquids of the same color. It has been taken in toxic doses as an abortifacient. In Martinique 4

[1] *Lancet*, 1883. [2] *Ibid.*, 1892 [3] *Ibid.*, 1887.
[4] Hall, *New York Med. Jour.*, 1883.

cases of homicidal poisoning were caused by it at the hands of one person.[1]

Symptoms.—When ferric chlorid has been given experimentally to the lower animals with food, it has been found harmless even in considerable doses. The same amounts given fasting and in alcoholic solution have resulted in death in from one to sixteen hours. It causes an inky, metallic taste, violent abdominal pain, vomiting, diarrhea, paralysis of the extremities, suppression of urine, convulsions, and death. The feces are blackened by the iron sulphid formed.

Fatal Dose.—One case has been reported of death after five weeks from a dose of the chlorid equal to an ounce and a half of the "tincture of iron." An ounce[2] has caused vomiting and urinary symptoms. On the other hand, a man[3] aged seventy-two recovered from the effects of three ounces of the tincture.

Treatment.—The alkaline bicarbonates or the carbonates dissolved in a large amount of water or milk may be swallowed or used to wash out the stomach with a pump or tube. The gastro-enteric symptoms should be treated by rest and anodynes.

Postmortem Appearances.—In one of the Martinique cases cited above a greenish-black, fur-like "mud" covered the tongue, esophagus, and stomach; swelling, congestion, and ecchymotic points were the changes noted in the liver and kidneys, and hyperemia marked the brain and its membranes.

Tests.—*Ammonium sulphid* causes a black precipitate of iron sulphid. It can be used after the metal has been extracted from the tissue with acetic acid. Redissolving the sulphid in nitrohydrochloric acid, the iron will yield to *potassium ferrocyanid* a blue precipitate. If the iron solution is almost neutralized with ammonia, then *ammonium sulphocyanid* will give a red color.

Detection.—Having digested the organic matters thoroughly in water acidulated with acetic acid, filtered, evaporated the filtrate to dryness, and incinerated the residue, the ash is treated with dilute sulphuric acid and the solution tested as above with ammonium sulphid and potassium ferrocyanid. Determination of poisonous amounts must rest upon the quantity found in the organs in excess of that normally present. The black fur on the mucous membranes and the stains on the clothing ought to yield significant amounts.

ZINC.

(Chemical Symbol, Zn.)

This is a bluish-white metal used in the arts in the form of sheet zinc, from which culinary vessels are made; also, under the name of "galvanized iron," as a plating on iron tubes or vessels. On exposure to moist air a white rust or film of oxid forms, which does not dissolve in water unless the water contains chlorids. The well-known fact that

[1] *Annales d'Hyg. Pub.*, 1879.
[2] Taylor, A. S., *Principles and Practice of Medical Jurisprudence*, 1883.
[3] Blyth, A. W., *Poisons, Effects and Detection.*

milk keeps sweet longer in zinc vessels than in pots is explained by the neutralization of lactic acid by the zinc, which is taken up as a lactate. Not only may milk thus be contaminated, but also vinegar, soup, olive oil, and alcoholic liquids. The symptoms produced by articles of food thus contaminated are not grave, and the effects of zinc oxid upon those who work in zinc factories are so inconspicuous as hardly to deserve the name of poisonous. The "zinc fever" is sometimes marked by indigestion, headache, colic, diarrhea, cramps in the legs, and peripheral neuritis, symptoms which might be attributable to the arsenic which is usually present in commercial zinc.

Poisonous Salts.—Of 65 cases of zinc-poisoning collected by Witthaus,[1] 46 were in Great Britain and only 3 in the United States. All were caused by two salts, the sulphate and the chlorid. The sulphate was to blame in 25 cases, of which 8 were due to mistaking it for "Epsom salt"; 5 were suicidal, and 4 were homicidal. Zinc chlorid was the poison in 40 cases, of which 27 were accidental and 7 suicidal. The form used in 26 cases was "Burnett's disinfectant"; in 4 it was "soldering fluid."

Zinc sulphate ($ZnSO_4 7H_2O$), or "white vitriol," is a freely soluble salt and occurs in crystals so closely resembling magnesium sulphate that it is often mistaken for it. The zinc salt is sometimes kept in the household as a prompt emetic for emergencies. "Epsom salt" is also a domestic remedy, and both are often kept in the same closet, in loose packages without labels.

These facts account for the frequency with which accidental poisoning occurs. In doses of twenty or thirty grains zinc sulphate will evacuate the stomach without causing much depression. This effect is so constant that even after doses of an ounce are taken recovery is the rule. When complete expulsion does not occur, it acts as a gastro-intestinal irritant, causing vomiting, purging, and, secondarily, dangerous prostration. In one case there was neither vomiting nor purging, but death occurred in less than four hours from the depressing action on the nervous system.

Zinc chlorid ($ZnCl_2$) is a very soluble, deliquescent salt, present in "Burnett's disinfectant," also in the "soft injection" fluid used for preserving bodies for dissection.

A soldering fluid is made extemporaneously by dissolving zinc to saturation in hydrochloric acid. This fluid is used to cleanse the surface of metals, so that the solder can make a perfect joint. In the shape of fused caustic sticks the chlorid is used to transfix cancerous tumors, the effect being to disorganize the growth for a considerable area, as the salt absorbs water from the tissues and diffuses readily. It is sometimes applied as a paste by cancer quacks in so careless a manner as to cause death. Blyth[2] mentions a fatal case in which this external application to the breast produced general symptoms of poisoning by zinc, and the metal was found in the liver and other organs.

Symptoms from Zinc Chlorid.—The gastro-intestinal symptoms

[1] Witthaus and Becker, *Medical Jurisprudence, Forensic Medicine, and Toxicology*, New York, 1896.
[2] Blyth, A. W., *Poisons, Effects and Detection*.

are those of a powerful corrosive—a metallic taste with instant burning pain in mouth, throat, and stomach. The act of swallowing is difficult and painful, and the salivary flow excessive. Violent vomiting begins immediately, often of bloody matters; purging supervenes, with tenesmus and bloody stools. Collapse may end in coma and death in a few hours. If life is prolonged, nervous sequelæ are common, such as perversion of the special senses, localized muscular spasms, muscular weakness, and aphonia. The local action may cause stricture of the gullet or pylorus, and also destruction of the glandular structure of the stomach, thus impairing digestion, so that inanition, extreme wasting, and even death may ensue.

Fatal Dose.—The prompt emetic action of zinc sulphate has brought about recovery after doses of an ounce; death has ensued from taking 1½ ounces. The caustic action of zinc chlorid has caused death secondarily after several weeks from the administration of 6 grains. Recovery has been brought about after a dose of 200 grains.

Fatal Period.—While death has occurred in about four hours[1] from administration of zinc sulphate without vomiting, and in another case from zinc chlorid, yet there are instances of death from the secondary effects of disorganization of the stomach and stricture of the gullet as late as one hundred and sixteen days after the dose.[2]

Treatment.—The efforts of the stomach at evacuation must be assisted by free drafts of warm water or warm milk. The stomach-tube may be used in the very exceptional cases when emesis is not prompt. The antidotes are milk, eggs, and the vegetable astringents containing tannin, represented by strong decoctions of green tea.

Postmortem Appearances.—The usual consequences of irritant poisoning, more or less intense, are to be seen—*i. e.*, congestion in the mouth, gullet, stomach, and intestines, areas of softening, ulceration, and even perforation. When death is due to secondary starvation, there is usually narrowing of the gullet, with thickening and corrugation. Jalland[3] reported a case of death on the eighty-eighth day, in which the stomach, as a digestive organ, was practically obliterated. It was found to be a part of a mass of organized inflammatory products matting together the omentum, pancreas, liver, spleen, and diaphragm.

Tests.—*1. Hydrogen Sulphid Test.*—A stream of this gas precipitates white zinc sulphid from an alkaline or neutral solution, or a solution made acid by acetic acid. This precipitate is soluble in the mineral acids, but insoluble in acetic acid, the alkalis, and the alkaline sulphids.

2. Ammonium sulphid gives the same precipitate, the only white insoluble sulphid obtained by this procedure.

3. Potassium ferrocyanid can be used to distinguish zinc sulphate from magnesium sulphate and oxalic acid, both of which have been mistaken for it. White zinc ferrocyanid is thrown down from a solution containing zinc sulphate, but the two others yield no precipitate.

Detection.—Organic matters supposed to contain zinc may be

[1] Penfeld, *Austral. Med. Jour.*, 1883. [2] Tuckwell, *Brit. Med. Jour.*, 1874. [3] *Ibid.*, 1887.

digested at a gentle heat with dilute acetic acid, filtered, the filtrate concentrated, and the metal thrown down as sulphid by a stream of hydrogen sulphid. This precipitate, collected on a filter, is washed, dissolved in strong nitric acid, evaporated to dryness, the residue taken up with water, and precipitated as a hydratocarbonate by adding sodium carbonate and boiling thoroughly. Having filtered and washed the precipitate, it can be dried, ignited, and weighed as ZnO.

A small portion of the hydratocarbonate may be fused on platinum with a drop of cobalt nitrate. The zinc is detected by a green color resulting.

METALS OF THE ALKALINE EARTHS.

BARIUM.

(Chemical Symbol, Ba.)

Certain salts of barium used in pyrotechny, in wood-staining, and in glass-making sometimes figure in toxicology as irritant poisons which, when absorbed, cause cardiac depression and convulsions. The *chlorid* and the *nitrate* occur in white, soluble crystals resembling the ordinary purgative "salts," for which they have been taken by mistake.

Symptoms.—Gastro-intestinal irritation is shown by vomiting and diarrhea, with straining and abdominal pain. After absorption dilatation of the pupils with convulsions, paralysis, and heart failure may supervene.

Fatal Dose.—About 100 grains (6.5 gm.) of the chlorid proved fatal to a woman, although, by gradually increasing the daily quantity, Pivondi was enabled to take in divided doses 119 grains (7.7 gm.) in one day.

Fatal Period.—Death occurred in one case in one hour, in another case in fifteen hours, in another case in thirty-four hours, and again as late as a week after taking the poison.

Treatment.—The best chemical antidote is magnesium sulphate (Epsom salts) or sodium sulphate (Glauber's salts). Both have the power to precipitate the barium as insoluble sulphate. The stomach should then be washed out with milk and water. Anodynes are indicated for the pain; heat and stimulants for the cardiac depression.

Postmortem Appearances.—Any or all of the signs of gastro-intestinal inflammation may be present—*i. e.*, patches of redness, swelling, softening, effusions, ulceration, and even perforation.

Linossier,[1] by experiments on rabbits, found that after chronic poisoning for thirty days all the organs contained barium—the bones most of all, the kidneys, brain, and spinal cord showed a less amount, the liver still less, and traces only were in the lungs, heart, and muscles.

Tests.—1. *Dilute sulphuric acid* precipitates barium sulphate, which is insoluble in hydrochloric or nitric acid.

2. *Neutral potassium chromate* gives a yellow precipitate, insoluble in water, but soluble in nitric or hydrochloric acid.

[1] *London Med. Record*, 1887.

3. A green hue is given to a colorless flame when a barium salt is held in it by a loop of platinum wire moistened with hydrochloric acid.

Detection.—Having dissolved the organic matter by hydrochloric acid and potassium chlorate and precipitated most of the common metals by hydrogen sulphid and ammonium sulphid, the filtrate may be tested for soluble barium salts by dilute sulphuric acid, or the organic matter may be burnt, the ash fused with sodium carbonate, dissolved in hydrochloric acid, and tested with sulphuric acid.

ALKALOIDAL POISONS.

ALKALOIDS are the main poisonous principles of plants. They include a large proportion of the known nerve poisons, and they act directly upon the nervous system, many of them acting through nerves upon certain muscles of the body. The several alkaloids are distinguished by the physiologic effect of each in its individual power upon different parts of the nervous system. The physiologic effects as obtained upon animals are limited according to the nervous organization in each species, and are more fully and surely obtained upon the developed nervous system of man.

The Chemical Character and Constitution of the Alkaloids.—In classification the alkaloids are basal, electropositive in their ability to unite with acids and produce salts. Strictly speaking, an "organic base" may be a union of carbon with any element of the nitrogen family and with hydrogen preponderating over oxygen, if the latter be present. Ammonia is the inorganic type of the organic nitrogen bases, as phosphin and arsin are types of like organic bases formed by phosphorus and by arsenic. Pyridin is the structural type of the principal poisonous vegetable alkaloids and of the alkaloids which have the most marked chemical character. The pyridin nucleus, holding an atom of nitrogen with five of carbon in a closed chain, has all the stability of the fundamental benzene type, and has, besides, a special capability of additive combination. However complex the alkaloid, its electropositive polarity centers in the pyridin-like ring and in its nitrogen member. In the solanaceous alkaloids and in those of coca the structure is simply that of pyridin extended in side or cross chains of carbon. In the quinolin nucleus, which enters into the strychnos alkaloids, the pyridin is reinforced by a conjoined benzene ring. In some of the opium alkaloids the three-ring structure of phenanthren is united to the nitrogen nucleus, which differs from that of pyridin in the admission of oxygen instead of carbon in one of the six positions of the cycle. These closed chains are all of the six-membered type. Vegetable alkaloids are their highly complex derivatives. Such are the deductions drawn from the actual data of synthesis and of analysis, and they accord with the obvious character of these alkaloids, their clearly marked properties, and the distinctness of their deportment in analysis. Molecular individuality appears alike in their physiologic power and in their chemical activities.

The chief alkaloids of the fungi and the animal alkaloids or leukomains, as well as the ptomains or bases due to bacteria, are for the most part constituted with open chains of carbon as nitrogen bases. In some of these bases, however, nitrogen is found in closed chains of other than the regular type of six-membered rings, such as indol, which is of the type of pyrról joined to benzene. Numbers of alkaloids, ptomains, and leukomains are as yet undetermined in chemical structure. Guareschi enumerates seventy ptomains and leukomains, among which twenty-six have been found to have open structure, and but seven have been found to agree with the simple closed chain structures of the pyridin type. Of the latter, an example is the dihydrotoluidin, secondary base, obtained by Gautier and Mourgues[1] from cod-liver oil. Putrescin and cadaverin[2] are open-chain diamins of very simple constitution. The alkaloids of tea and the other beverage plants are diureids, now represented as derivatives of purin.

The element oxygen is found in the non-volatile alkaloids, and the structural relations of this element give the key to several peculiarities. Hydroxyl, especially if phenolic, gives solubility in the alkaloids. Ester formations, as in atropin and in a two-fold way in cocain, render an alkaloid saponifiable[3] as truly as a fat.

Identification in Analysis.—In respect to the bearing of structural chemistry upon fallacies of analysis in identification of the poisonous vegetable alkaloids, it is a reasonable conclusion that compounds of such remarkable chemical individuality ought to be identified by the analyst, and that he ought to be able to distinguish a vegetable alkaloid from a ptomain, at all events quite as surely as he can distinguish one vegetable alkaloid from another. Now when greater numbers of bases of vegetable and of putrefactive origin are becoming known, the danger of mistaking one for another is seen to have been greater than has been realized. But in this advance of chemical knowledge the resources of analysis are being enlarged and enriched as well. With these resources, when they are faithfully employed, greater precision is attainable in the determination of minute quantities of poisons, such as are recovered in postmortem analysis. Meanwhile the inherent limitations upon this recovery are more definitely understood. The new light shed upon the constitution of the organic bases reveals new means of identification and gives meaning to the detail of chemical tests.

Upon the important question of mistaking ptomains for vegetable alkaloids the conclusions of Guareschi, as given in his admirable work on this subject,[4] are presented in full in the following paragraphs:

[1] *Compt. rend.*, 1888, pp. 107, 110, 254.
[2] Vaughan and Novy, *Cellular Toxins*, 1902, pp. 267-273.
[3] Prescott, *Organic Analysis*, 1888, p. 170.
[4] *Alkaloids mit besonderer Beruchsichtigung der vegetabilischen Alkaloide und der Ptomaine*, von Dr Icilio Guareschi, Professor an der konigl. Universität Turin. Dr. Hermann Kunz-Krause, Berlin, 1896. See pages 597 and 600. Guareschi and Mosso, "Les Ptomaines, Recherches chimique, physiologiques et médico-légales," *Arch. Ital. de Biologie.* Guareschi, 1887, *Gaz. chim. ital.*, vol xvii., p. 503.

Similarities of ptomains to plant bases make it very probable that before the researches of Selmi ptomains have been frequently mistaken for alkaloids in medicolegal cases. This conjecture increases in probability from the fact that formerly many chemists considered it to be sufficiently conclusive of poisoning to have extracted from the already putrefied intestines a poisonous alkaline-reacting substance giving all the general reactions of the alkaloids. To-day it is not so difficult to avoid that fallacy, although the discovery of cadaver alkaloids places a considerably higher responsibility not only upon the judgment but also on the knowledge and experience, of the chemist. On the other hand, the chemist of to-day must guard against the other extreme, that of allowing the vegetable alkaloids to leave the field altogether to the ptomains—attributing to the presence of the latter observed reactions which really are those of plant alkaloids present in the organism. All vegetable alkaloids, with a few exceptions, can be characterized as such by their chemical and physiologic behavior, so as to be recognized in the presence of ptomains. A careful and experienced worker of to-day will not confound a vegetable alkaloid with a ptomain that is known and worked out, since, in spite of the great number of the ptomains, none has been observed to give reactions uniform in all points with any plant base.

An exception to this general statement may perhaps be made in case of poisoning occasioned by muscarin, and certain actively poisonous bases of the pyridin and hydropyridin series, as the last-named bases also occur among products of putrefaction. In these cases the proof of the occurrence of poisoning from analytic results alone is not only very difficult, but in the majority of cases impossible.

Moreover, special caution in expression of opinion is needful in all cases where the amount of the vegetable alkaloid extracted from portions of the body is too small to yield all the characteristic chemical and physiologic reactions. The same is true when the alkaloid cannot be obtained in purity, but remains mixed with ptomains and extractive matters. In all these cases it is practically impossible to draw definite and unobjectionable conclusions from analytic results.

Another consideration especially important from a chemicolegal point of view is the possibility of changes taking place in the composition of alkaloids under analytic treatment, such as those resulting in so-called amorphous alkaloids, some of which are insufficiently examined. The possibility of such changes makes it the strongest duty of the chemist fully to find his bearings upon the properties and reactions of all such plant-bases as colchicin, delphinin, cannabin, oleandrin, pseudo-curarin, lobelin, the alkaloids of hops, and others which are known only in the amorphous condition (Guareschi).

Perhaps a not unseasonable illustration of what has been said is given in the following: In a chemicolegal investigation Brouardel, Ogier, and Pouchet apparently isolated a basic compound which fully agreed with colchicin in all its reactions, and besides its action seemed

to be indicated by the observed symptoms of poisoning and the post-mortem appearances. Nevertheless the chemists did not think these results sufficient to establish its identity, since there was the possibility that in the course of time ptomains as yet unknown would be discovered which in their chemical reactions agree with colchicin. The testimony furnished by the defendant, and brought out by Schutzenberger and Vulpian, partially corroborated the results of the first investigation. The last-named experts, in similar analytic work upon two cadavers in which the possibility of poisoning by colchicin was precluded, succeeded in isolating a ptomain which coincided in its reactions with the base isolated from the suspected organs, and also showed reactions very similar if not identical with colchicin. The outcome of these statements was the acquittal of the defendant. But if the research of Zeisel (unfolding the constitution of colchicin) had been known at the time, perhaps the result of that forensic inquiry might have been reversed.

Properties of Vegetable Alkaloids.—In respect to physical state these substances may be divided into: (1) Non-volatile alkaloids: very numerous, composed of carbon, hydrogen, nitrogen, and oxygen, colorless or white solids, melting when heated and usually subliming with partial decomposition, but inodorous and incapable of distillation unchanged. (2) Volatile alkaloids: comparatively few in number, composed of carbon, hydrogen, and nitrogen, without oxygen, for the most part liquid when free as bases, abundantly odorous and capable of distillation unchanged, even at ordinary atmospheric pressures. Nicotin, the odorous and poisonous principle of tobacco, is an example of the second division, as quinin is of the first division.

Both classes are capable of neutralizing ordinary acids, with the production of salts of the alkaloids, such as a sulphate or hydrochlorid.

Solubilities.—*In Water.*—When free from combination with acids, the greater number of vegetable alkaloids are counted as insoluble in water—that is, their solubility in water is very slight. Thus, it is stated that strychnin is soluble in 6700 parts of water at 15° C. (59° F.) or in 2500 parts of boiling water. Cocain is reported to dissolve in 1300 parts of cold water. On the other hand, the ordinary salts of alkaloids, such as the sulphate, nitrate, acetate, or hydrochlorid, dissolve quite freely in water. The solutions are neutral to litmus and most other test-papers. Strychnin sulphate requires but about 40 times its weight of cold water to dissolve it; morphin sulphate requiring about half as much of the same solvent. It is in correspondence with the facts just stated that in ordinary aqueous solutions of the salts of the alkaloids the addition of any caustic alkali (short of excess) will cause an immediate precipitation of the uncombined alkaloid. Excess of the caustic alkali will redissolve such precipitates of alkaloids in some instances, but not in others, so that the effect of an excess of alkali is a means of distinguishing alkaloids from each other, a reaction seldom delicate enough to be useful with very small quantities. The effect of alkalis upon the ester-

like composition of the so-called saponifiable alkaloids is not an immediate effect, and, though a reaction to be regarded, it is one likely to escape observation unless made a subject of inquiry.

Solubility in Alcohol.—In general, both the free alkaloids and their salts are soluble in ordinary alcohol with considerable abundance, and in proportions varying with the strength of the alcohol. Alkaloidal salts usually dissolve in alcohol more abundantly than do the free alkaloids; therefore a partial precipitation of alkaloid is obtained in some cases by adding alkali to alcoholic concentrated solution of alkaloidal salt, but this reaction is uncertain. Absolute alcohol is valuable as a solvent to separate alkaloids from various tissue-substances. This separation from proteid and other matters requires to be made gradually with increasing strengths of the alcohol, or else very thorough washing of the finely divided precipitate, to avoid waste of the alkaloid by its retention in the matters coagulated by the alcohol.

Ether and Chloroform as Solvents.—These are the best known of the so-called immiscible solvents, those not miscible with water and separating from aqueous solutions in which they have been mixed by shaking. Neither chloroform nor ether, however, is entirely immiscible with water. The free alkaloids differ from each other as to solubility in ether and in chloroform, and these solvents have been somewhat used as a means of separating alkaloids from each other. The scheme introduced by Dragendorff in 1867 is the most elaborate plan for such separation, five or six immiscible solvents being used.[1] But these solvents are more in use to effect a separation of whatever alkaloid be in hand from matter other than alkaloids, which is also done in Dragendorff's process; and in this use the analyst avails himself especially of this fact, that the sulphates, hydrochlorids, and other salts of the alkaloids refuse to dissolve in the immiscible solvents with a very few well-known exceptions. This fact was first made available by Otto in 1856, in his modification of the method of Stas for the extraction of alkaloids in toxicology. For example, from an acidulous aqueous solution containing strychnin, chloroform will dissolve out and remove whatever matters are soluble in this solvent, leaving the alkaloid as a salt in the watery solution. Now the liquid is made alkaline and treated with the chloroform, in which the free alkaloid dissolves, and a distinct step in purification is taken. By the repetition of these stages of treatment the alkaloid is gradually purified. In the complex scheme of Dragendorff, already mentioned, all his immiscible solvents are employed successively in acid and then in alkaline solution, the aqueous liquid being shaken out with each solvent, which is left to separate and then drawn or siphoned off. Ether and chloroform are used also upon solid residues, stirring to dissolve, as well as in an "extraction apparatus" and in other ways in the course of analytic work.

Other Immiscible Solvents.—The benzene (benzol) of coal-tar distillation, petroleum ether, amyl-alcohol, acetic ether, acetone, are used in the

[1] For description of the Dragendorff process see p. 331.

same general ways stated for ether and chloroform, according to the adaptation of each.

General Reagents for the Precipitation of Alkaloids.—Alkaloids agree in promptly forming certain inorganic combinations, such as double salts and perhalids, and a few organic combinations, all insoluble in water, so that they are instantly thrown down from aqueous solutions of alkaloidal salts on adding the proper reagent.

1. **Iodin in Solution of Potassium Iodid (Wagner's Reagent).**[1]—A decinormal solution of iodin with sufficient potassium iodid. For qualitative uses about 1 part of iodin and 2 parts of potassium iodid in water to make 100 parts of solution. Applied in acidulated solutions, not alcoholic, and added in excess. The precipitates, of varying shades of dark brown to black color, are periodids. The higher periodids, by excess of reagent, are more stable than the lower periodids.[2] By action of sodium thiosulphate solution the alkaloidal precipitate is dissolved, the periodid being reduced to a hydriodid. When the latter is made alkaline and treated with an immiscible solvent, the pure alkaloid is recovered.

2. **Potassium Mercuric Iodid Solution (Mayer's Reagent).**—For qualitative use by adding to solution of mercuric chlorid a little more than enough of solution of potassium iodid to dissolve the colored precipitate at first formed. This reagent is to be invariably applied in an acidulous solution, and in absence of alcohol or acetic acid. The precipitates are yellowish-white in color. On treating the precipitate with stannous chlorid, then adding caustic potash to alkaline reaction and extracting with an immiscible solvent, the free alkaloid is obtained.

3. **Phosphomolybdic Acid.**—A sodium or potassium (not ammonium) salt of phosphomolybdic acid dissolved with the help of strong nitric acid, and used in this acidulous solution. This reagent is a close precipitant of the alkaloids. If ammonia be added to the precipitates, in case of certain alkaloids having a reducing (deoxidizing) action, the precipitate turns blue or dissolves with a blue color.

4. **Bromin** dissolved (to saturation) in aqueous hydrobromic acid. A close precipitant of most alkaloids, a favorite reagent of the late Professor Wormley, especially for microchemical work.

5. **Tannic acid,** in freshly prepared aqueous solution, precipitates the alkaloids with more or less completeness, the precipitate being in some cases prevented or dissolved by acidulation with mineral acids.

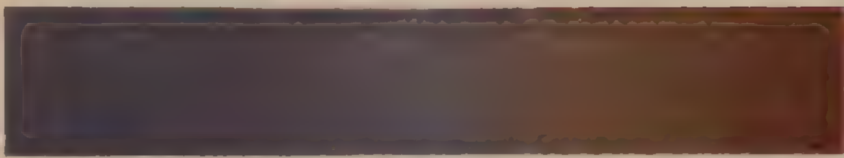
6. **Picric acid** in water solution gives beautiful precipitates, many of them crystallizable, with a number of the alkaloids.

Use of General Reagents for Negative Tests.—There are various substances not alkaloids precipitated from their aqueous solu-

[1] Bouchardat, *Compt. rend.*, 1839, vol. ix., p. 475; R Wagner, *Zeitschr. anal. Chem.*, 1861, vol. i., p. 102.

[2] Prescott and Gordin, *Jour. Amer. Chem. Soc.*, 1898, vol. xx., p. 709; *Proc. Amer. Phar. Assoc.*, vol. xlvii., p. 357.

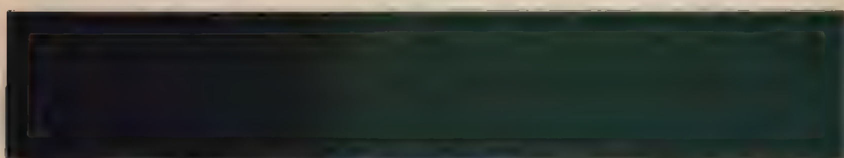
PLATE 7.



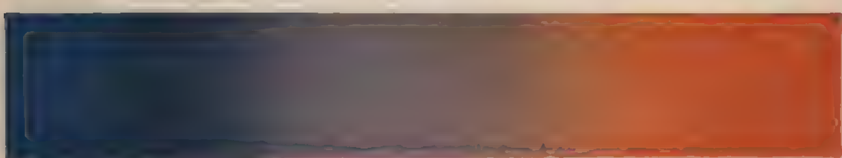
Atropin with Vitali's test.

Brucin with nitric acid followed by stannous chlorid.

Mixed alkaloids of gelsemium with sulphuric acid and ceroso-ceric oxid.



Morphin with ferric chlorid.



Strychnin with sulphuric acid and ceroso-ceric oxid, or manganese dioxid.

Veratrin (mixed alkaloids) with sulphuric acid.

ALKALOIDAL COLOR REACTIONS.

NOTE.—It is practically impossible to imitate by the lithographic art every shade of color produced in alkaloidal reactions, and consequently, while the above chart gives an excellent idea of the colors obtained, they are not in every respect absolutely correct.

tions by the general reagents just described. The final solution obtained in the processes of extraction of alkaloids from the tissues of the body, though carefully purified by action of alcohol and of immiscible solvents from any remaining traces of ordinary proteid matters, will generally give a slight turbidity, due perhaps to leukomains, under action of potassium mercuric iodid or of phosphomolybdic acid, and sometimes under action of iodin. If a quite concentrated solution so obtained give only a slight turbidity with such a general reagent, this result may be accepted as negative for the presence of a poisonous alkaloid, unless in too minute a quantity to be precipitated. It will be usually advisable to apply such special chemical or physiologic tests as are of greater delicacy than the tests by general reagents.

More decided precipitation by a general reagent, applied to a well purified extract from tissues or organs of the body, certainly calls upon the analyst to apply special tests for poisonous vegetable alkaloids with the utmost care and with economy of material. But such is the liability of precipitation of ptomains or of leukomains in unusual abundance under action of the general reagents, and such the possibility that some kind of "animal extractive matter" besides the bases, possibly approaching a proteid composition, may have escaped removal in the processes of purification, that even a considerable precipitate by a general reagent cannot be taken as conclusive of the presence of a vegetable alkaloid, still less of a poisonous one. In analyses of food materials or medicines a precipitate by a general reagent has more positive bearing than in the analysis of tissues, but even here the analyst must not base very much upon the reaction of a general reagent alone.

Special Chemical Tests and Color Reactions.—The most satisfactory of these, as tests for the chief alkaloidal poisons, are given in the pages following in this section. Great numbers of other special tests have been and are being reported for the identification of alkaloidal poisons. In the application of any such test the analyst must rely chiefly upon his own observation and experience of the reaction with a known sample of the alkaloid, taken in such minute quantity as would best represent what might be recovered in analysis. To cover questions of interference, through extraction from tissues, the test may well be applied to what is recovered after adding the known alkaloid to some tissue material and conducting the extraction parallel with that of the analysis itself.

Physiologic Tests.—Examples of these and their most important uses will be found in the text for strychnin, atropin, and aconitin. Their relative value is stated in each case. Each test, chemical or physiologic, is to be valued upon its merits, judged from all known data in the experience of chemists, pharmacologists, and practical analysts. It cannot be said that chemical evidence is insufficient without physiologic data in the examination for all poisonous alkaloids. In analysis for aconitin the physiologic test is the main reliance; in search for atropin it is perhaps the best dependence; in the work for strych-

nin it is strong confirmation; in examining for cocain it is one of several tests; while among the means of identification of several other alkaloidal poisons physiologic methods have only a subordinate place.

The delicacy of tests for alkaloids as a class is not exceeded by the delicacy of tests for inorganic poisons. The least quantity of an alkaloidal poison which is sufficient for its positive identification when it is taken by itself in well-known tests is very small when compared with poisonous doses, or, in general, even with medicinal doses. The chief task of the analyst, however, is to obtain the poison by itself through its removal from other matters.

Separation from Animal Tissues.—Notwithstanding the peculiar individuality of the vegetable alkaloids, shown both in their physiologic effects and their chemical constitution, already remarked upon on page 447, they still share in the frailty of all organic compounds. Their separation, in amount comparatively minute, from overwhelming masses of organic matter in tissues and foods, is a task far more delicate than the extraction of inorganic poisons. No method which involves the corrosion or chemical alteration of tissue-substances would be possible. All the processes are dependent upon the action of solvents and of reagents which convert the alkaloids into their salts or other combinations, or else liberate them from like combinations previously formed. Under the head of Solubilities (page 450), also of General Reagents (page 452), the principles are stated upon which the various methods of separation of alkaloids from tissues have been elaborated.

Directions for the Separation of Alkaloids from Tissues.[1]—These are given in different places in the text with the purpose to adapt the process to the character of the alkaloid, as follows:

Under Morphin.—A process advisory for an alkaloid that must be especially guarded against oxidation.

Under Atropin.—The treatment recommended when the alkaloid must be guarded against hydrolysis (saponification). Applicable to cocain, and with additional precautions to aconitin, as specified.

Under Strychnin.—A process proposed when the alkaloid is to be guarded against contamination with remaining impurities, and will bear treatment of comparative severity for separation. The treatment with hot concentrated sulphuric acid, however, is admissible with no other alkaloid than strychnin.

Under Nicotin.—A method for a volatile alkaloid, to be guarded against waste by vaporization.

Chemists of experience in the work will very properly adopt such outlines of methods as have been found by them to be most satisfactory and secure. The principles of separation are to be ever borne in mind. The skilful analyst will modify whatever outline he uses so as to accomplish separation and purification gradually, step by step, prudently guarding against waste, as required in quantitative work.

Analyses for Alkaloidal Poisoning Before Death.—In cases

[1] Consult also p. 330 *et seq.* in section on General Principles of Toxicology.

of patients under treatment, when poisoning with an alkaloid is suspected, the urine should be subjected to strict analysis for the poison or securely retained for such analysis, as should also the vomit or washings of the stomach if they can be obtained. If aconite poisoning be suspected, the saliva likewise may be analyzed. (See the text under the head of Deposition in the Body for the several poisons, and Atropin, Case 22; Morphin, Case 23; Strychnin, Cases 14, 18, 20.) Portions of food or of medicine under a reasonable suspicion of being poisoned should also be retained under seal.

A Control Analysis.—If the operator would ascertain just what is really accomplished by a given process of separation from tissues, or by the process he has decided to use, he can do no better than to conduct, at the same time, and in all respects with the same conditions, the given process upon the same quantity of like tissues, in about the same stage of decomposition, in which a weighed quantity of the poison in question has been intimately mixed. The quantity of the poison should be minute enough by careful calculation not to be in excess of what would be present in a real case of poisoning. It must be recognized that a brief mechanical mixture of the dissolved alkaloid with the comminuted tissue material or other organic mass will not ensure as great a difficulty of separation as that which is met with in a case of poisoning. Therefore the records of results of analysis of the remains of poisoned animals are of material advantage; and experiments where the tissue-substance has been charged with a known (minute) quantity of the poison, either by administration to the animal before death or by intimate admixture to dead tissues, and the whole then allowed to putrefy, are therefore more conclusive in their bearing upon analyses made some weeks or months after death. In such cases the experimental putrefaction should be conducted in the same conditions of exclusion or limited access of air, and of temperature as near as can be, as those of the burial of the remains under investigation.

Extraction of Alkaloids from Tissues not Without Inherent Waste.—This has been proved by experiment. It may be understood from the laws of physics. Solubility and insolubility are but relative terms which we use for differences of degree. A coagulum or undissolved residue will retain by adhesion some fraction of what is dissolved in the filtrate, however this fraction may be divided by washings. There is "analytic error" even in inorganic analyses, and when dealing with colloidal matter in masses, several tens of thousands times larger than the crystalloidal poisons to be recovered, the unavoidable "analytic error" may exceed the ratio of the quantity of the poison, or leave less than what is needful for its identification.[1]

[1] "Control Analyses and Limits of Recovery in Chemical Separations," A. B. Prescott, *Chem. News*, London, 1885, vol. liii., p. 78. In the systematic execution of a given process of separation of strychnin from muscular tissue, with 50 gm. of the latter, the loss of the alkaloid was about 0.0004 gm. Therefore, in separating strychnin from a pound of the tissues, about 3½ milligrams of the alkaloid were lost. This makes the waste of the alkaloid, in separating from one pound of tissues, over one thousand times the least quantity necessary for identification when the alkaloid is taken alone in a qualitative test.

Results of Experimental Analysis for Alkaloids.—In experiments with the writer,[1] in 1883, Mr. G. A. Kirchmaier found that strychnin was not uniformly recovered, even to a qualitative extent, from the blood of animals poisoned with it, nor from organs of the body. From the blood of the heart and of the general circulation the poison was obtained by analysis in two cases out of four, $\frac{1}{8}$ to $\frac{1}{4}$ gr. (0.008 to 0.016 gm.) of the alkaloid having been administered hypodermically to cats two to eight minutes before death and the examination commenced half an hour after death. From the liver a trace of the poison was recovered in only one out of six of the same series of experiments, one in which $\frac{1}{8}$ gr. (0.008 gm.) of the alkaloid was injected five minutes before death and analysis commenced half an hour after death. The kidneys were analyzed in the six experiments with a negative result. When $\frac{1}{32}$ gr. (0.002 gm.) of the alkaloid was introduced into the stomach of the animal eleven minutes before death, twelve hours afterward analysis of the stomach gave evidence of the poison. But when $\frac{1}{60}$ gr. (0.001 gm.) of the alkaloid was given by the stomach twenty minutes before death, analysis of this organ immediately after death yielded no indication of the poison.

Further on this subject, see Cases 14 to 20 under Strychnin; Case 22 under Atropin; Cases 23, 67, and Analyses 1 to 7 under Morphin.

Experiments to Guard Against Fallacies in Analysis.—These may be brought to bear upon a given qualitative test or a given method of separation from tissues, or upon both.

Nine portions of putrefied matter, representing as many organs of an animal body buried forty-five days in a container, "a tight wooden box," in March and April, were extracted first by alcohol acidulated with acetic acid, later with hot amyl-alcohol, along with other treatment, and the final extracts, as prepared for qualitative reactions, in no cases gave any indication of morphin by Lefort's test, made by iodic acid followed by ammonia. In all the cases, however, the ordinary test with iodic acid and starch gave the blue color of liberated iodin.[2]

To find at the same time the trustworthiness of Lefort's and Froehde's qualitative tests, and of the method of extraction from tissues presented under the head of Morphin, and to determine the liability of interference from products of tissue putrefaction when air is excluded, the following procedure was instituted: About $2\frac{1}{2}$ kilograms of finely chopped ox-liver was placed in a large bottle (*a*) securely stoppered with a perforated cork connected with a bent glass tube. The cork was sealed with paraffin and the outer end of the glass tube was allowed to dip into a cistern of mercury, thus excluding all communication with the outside air. Putrefaction was now allowed to go on, in a warm room, for about fifty days. Gas was given out during the earlier part of the decomposition. Into another bottle (*b*) another $2\frac{1}{2}$ kilograms of finely chopped ox-liver was placed with 0.52 gm. of morphin intermixed,

[1] *Contributions, Chem. Lab.*, University of Michigan, vol. ii., p. 91.

[2] D. L. Davoll, from work done in consultation with the writer, University of Michigan, 1894. *Jour. Amer. Chem. Soc.*, vol. xvi., pp. 805–807.

about 1 part in 5000, and this bottle was sealed, connected, and set aside with bottle (*a*) for putrefaction thirty-five days. The contents of bottle (*a*) in duplicate portions, in one with the process of extraction detailed under Morphin, and in another portion with Kippenberger's proposed extraction method,[1] gave final residues that, under the Lefort test by iodic acid, also under Froehde's reagent, in each portion brought absolutely negative results. On the other hand, the contents of bottle (*b*), that containing morphin when set aside, in two portions, with the first above specified extraction method, in a parallel Lefort test by iodic acid, gave a clear mahogany color, a strong indication for morphin. No indication of the alkaloid was gained from the extraction process of Kippenberger.[2]

Again in 1896, to prosecute the same inquiry under conditions of actual animal poisoning, when the poison is physiologically distributed among the tissues, with putrefactive changes in confinement from the air after death, Mr. H. T. Smith and the writer proceeded as follows: Of morphin 23 grains (1.5 gm.) were given through the mouth to a dog of about 15 kilograms weight; two hours afterward the animal was killed with an anesthetic, the body placed in a box made of sheet-iron, and the box sealed hermetically, this being on February 20. In the following month another dog of about 30 kilograms was killed with an anesthetic without any administration of morphin, and the body sealed up the same as the other one. The two boxes were buried on March 20, in a wooden case, under about three feet of soil. On the 8th of May, after about seven weeks' burial and about ten and a half weeks of inclosure of the body containing morphin, the two boxes were exhumed. The box containing the larger body, the one sealed up later, had burst from gas pressure within and its contents were in a state of decomposition more advanced than that of the smaller body. From each body the stomach, intestines, and liver were taken together in analysis by the method given under Morphin for separation from tissues, and also by the process proposed by Kippenberger. In the final result each one of the putrefied extracts from the body of the animal poisoned with morphin gave distinct reactions for morphin in Froehde's test and in Lefort's test, but gave no reaction with the ferric-chlorid test nor with sulphuric and nitric acids. The purified extracts from the body of the animal not poisoned, in parallel portions, gave no reactions for morphin in the tests just named. With Kippenberger's process the final residues failed to yield any reaction for morphin from the body of the poisoned dog, and gave no appearance of a morphin reaction with any of these tests in the extracts from the body not poisoned.[3]

Precautions Against Impurities in Reagents.—In addition to the proper tests of purity, to which the analytic chemist must subject all his reagents and solvents before taking them into use, advantage

[1] For description of the Kippenberger process see page 338, in section on General Principles of Toxicology.

[2] H. T. Smith, 1894, in work under the writer's observation, University of Michigan.

[3] Compare section on Ptomains and Other Bacterial Products in Their Relation to Toxicology.

can be taken of a blank analysis for control to exclude fallacious results from any source, and misleading reactions in the final tests. At any rate it is necessary to be assured that the immiscible solvents yield no residues that can react with the agents employed in testing for alkaloids, and that the residues of all solvents and reagents together give negative results in the tests that are depended upon.

Period of Detection of Alkaloidal Poisons.—The alkaloids present much variation among each other in their power to resist decomposition during the putrefaction of tissues in which they are deposited. No alkaloid can be affirmed to be indestructible under these conditions. Probably the resisting power of alkaloids has been underrated, owing to defective analyses. The records of analysis in actual cases, the data upon which we must mainly depend, are given under the proper subhead for each alkaloid.

ACONITE AND ACONITIN.

Aconitin represents the poisonous alkaloids of the drug aconite. The drug consists of the root, sometimes the leaf, of *Aconitum napellus* (monkshood) and other species of Aconitum. Aconitin itself, of the ascribed formula $C_{33}H_{45}NO_{12}$, is one of the most concentrated poisons known, and at the same time is a very unstable alkaloid, subject to changes under chemical treatment or by keeping, these changes being in the nature of conversion into other alkaloids of a lower degree of the same poisonous power, some of them being practically inert. These weaker aconite alkaloids make up a variable part of the total alkaloid of the drug and its liquid preparations, and a still larger part of many lots of so-called aconitin alkaloid on the market. The liquid preparations of the drug, chiefly of the root, are more widely used and more relied upon in medicine than is the separate alkaloid, and the latter has an uncertain place in national pharmacopeias. The total alkaloidal content of the good root is said to average from 0.75 to 1 per cent. The percentage of absolute aconitin in the root cannot well be declared from the results of analysis, but from data of physiologic effects it would appear that the poisonous power of aconite root is equivalent to not more than 0.01 to 0.02 per cent. of absolute aconitin. Wright obtained 0.03 per cent. and Juergens 0.02 per cent. of pure aconitin, besides alkaloid of lower power, from the root by analysis.

Aconitin proper is crystallizable—indeed, is somewhat loosely termed the crystallizable alkaloid of aconite, in contradistinction to the amorphous aconite alkaloids, which have little poisonous effect. In taste the bitterness of aconite is due to its alkaloids of less poisonous effect. Certain inert aconite products are very bitter.

Symptoms of Poisoning by Aconite.—In medicinal overdoses there are diminished force and frequency of pulse, coldness and moisture of the surface, tingling and numbness about the mouth, face, and throat, the urine being often increased. In decidedly poisonous doses there are tingling and numbness in the mouth, burning in the throat and stomach, flow of saliva, nausea, rarely purging, grinding of the teeth,

numbness and tingling of the fingers and other parts of the body, pain in the eyes and head, and difficulty in swallowing and sometimes in speaking. The pulse from the first is slow, feeble, and irregular, the surface cold and moist, the face bloodless, and the strength prostrated. Delirium is unusual, and stupor never appears as an effect of this poison. Respiration becomes slow as the case progresses. The state of the pupils varies in different stages and cases. In cases running a rapid course the symptoms are sometimes those of general shock.

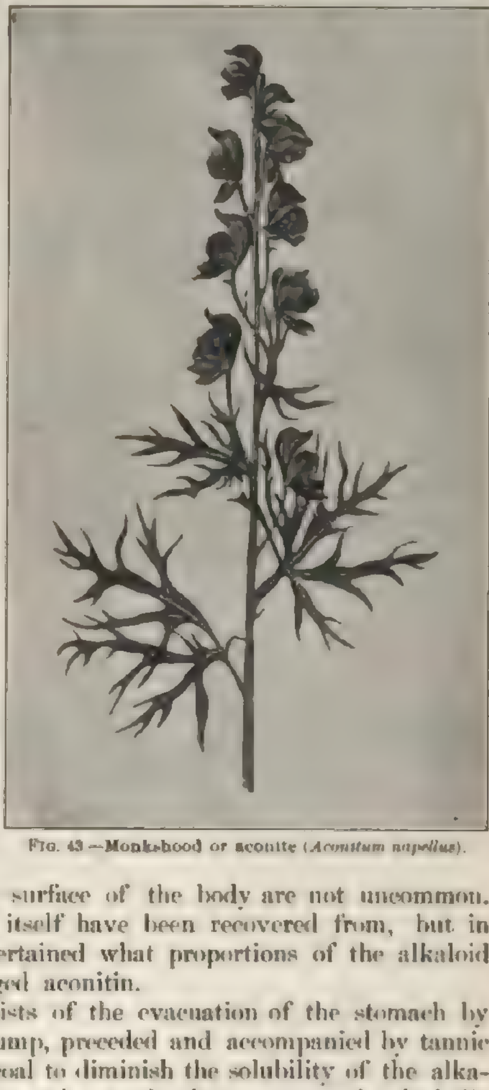


FIG. 43.—Monkshood or aconite (*Aconitum napellus*).

Period when Fatal.—The symptoms come on very soon—almost as soon as the poison reaches the circulation. The course is rapid. Death generally takes place in three or four hours. In one series of six fatal cases reported by Professor Mallet in 1883 the shortest fatal period was eight minutes and the longest was four days.

Fatal Quantity.—Pure aconitin is one of the two or three most powerful known poisons. It is stated (see Case 3) that $\frac{1}{16}$ of a grain (0.004 gm.) has caused death, and that one-half of this might prove fatal. Experiments indicate that $\frac{1}{32}$ of a grain (0.002 gm.) is about the least fatal dose for man. Cases of poisoning by absorption of aconite from the surface of the body are not uncommon. Large doses of the alkaloid itself have been recovered from, but in these cases it cannot be ascertained what proportions of the alkaloid taken were absolute unchanged aconitin.

Treatment.—This consists of the evacuation of the stomach by the siphon-tube or stomach-pump, preceded and accompanied by tannic acid or finely powdered charcoal to diminish the solubility of the alkaloid. Then the remedies known as heart-stimulants—ammonia, alcoholic drinks, and, with observant care, the hypodermic administration of digitalis and perhaps strychnin—are indicated. The patient should be kept warm (see Cases 2, 4, 5).

CASES OF POISONING BY ACONITE AND ACONITIN.

CASE 1.—One ounce (28 gms.) of aconite was taken by a woman forty-eight years of age. There was general collapse, the extremities were cold and clammy, and the breathing was labored and slow. The stomach was washed out and ether given hypodermically. The patient died in sixty-five minutes.[1]

CASE 2.—A dram (3.5 c.c.) of Fleming's tincture of aconite, which should have been equivalent to 306 grains (20 gm.) of aconite root, was taken by a man fifty years of age. The surface of the body was cold and moist, the pulse 96, and the pupils were slightly dilated. There was vomiting. Digitalis was given hypodermically, with alcoholic drinks, followed by calomel. The patient recovered.[2]

CASE 3.—Of crystallized aconitin nitrate (Petit's) $\frac{1}{150}$ of a grain (0.0004 gm.) was taken by a feeble man sixty-one years of age, who immediately suffered from burning and constriction of the mouth extending to the stomach, and a sense of coldness of the body. The patient took repeated doses within two days, amounting in all to about $\frac{1}{7}$ of a grain (0.0092 gm.) of the same aconitin nitrate, and suffered from violent symptoms of aconite poisoning, so that at one time it was believed that he would die; nevertheless he recovered. From the same solution of the alkaloid the physician who had prescribed it in the case just stated himself took about $\frac{1}{16}$ of a grain (0.004 gm.) of the aconitin nitrate. Symptoms of poisoning appeared at the end of fifteen minutes. There were burning in the mouth, extending to the abdomen, pallor, feebleness of the extremities, a small and irregular pulse, the pupils being at first contracted and then suddenly dilated. Vomiting was induced, and ether given hypodermically. Later there were convulsions, with labored respiration. The heart failed and the patient died five hours after taking the poison.[3]

CASE 4.—Four teaspoonfuls of tincture of aconite were taken by a man sixty years of age. Tingling in the face, facial muscles drawn, intense coldness, weak pulse, heavy breathing, and blindness followed. Under treatment with emetics, tincture of digitalis, wine, brandy, and strychnin the patient recovered.[4]

CASE 5.—A large amount of aconite was taken by a woman thirty-six years of age who had previously taken a large amount of alcoholic liquor. The symptoms were very much delayed. Treated hypodermically with digitalis, the patient recovered.[5]

CASE 6.—In a case of poisoning by 1½ ounces (45 c.c.) of Fleming's tincture of aconite, with numbness, profuse sweating, dilated pupils, general convulsions, and finally coma, treatment by emetics, brandy, digitalis, ether, and caffein was instituted and the patient recovered.[6]

CASE 7.—In 1883 Professor J. W. Mallet readily detected aconitin in the contents of the stomach of 3 out of 6 cases of fatal poisoning by aconite at the Western Asylum for the Insane in Virginia. In some manner the poison had been introduced into the medicine of 8 patients, 2 of whom recovered. Aconitin was found in the residues of the medicine taken.

CASE 8.—A man died from aconite poisoning five hours after the drug was taken. At the autopsy a paleness of the muscles was observed, the lungs were hyperemic, and the blood was thin and bright red.

Postmortem Appearances.—These are not markedly characteristic. The lungs, liver, and brain are generally found somewhat congested. The stomach is sometimes reddened. The blood is found abnormally fluid, and sometimes of a bright-red color.

Chemical Tests for Aconitin.—In cases of poisoning by the liquid preparations of aconite the poisonous alkaloid itself is the only aconite constituent capable of being recovered and identified in analysis.

[1] S. McWhannell, *Brit. Med. Jour.*, 1890, vol. ii., p. 791. [2] *Ibid., loc. cit.*

[3] A Busscher, *Berlin. klin. Wochenschr.*, June, 1880, p. 337; *Ann. d'Hyg.*, January, 1882, p. 87; P. C. Plugge, *Arch. d. Pharm.*, 1882; Wormley's *Microchemistry of Poisons*, 1885, p. 620.

[4] M. A. Warriner, *Med. Record*, New York, 1891, vol. xxxix., p. 521.

[5] C. F. Dercum, *Med. and Surg. Reporter*, 1889, Philadelphia, vol. lxi., p. 376.

[6] C. C. Bradley, *Med. Record*, New York, 1887, vol. xxxii., p. 155.

As already stated, aconitin is an unstable alkaloid, a fact that adds to other difficulties in its recovery and identification. The alkaloid readily undergoes hydrolysis or saponification, even by boiling with water, yielding, among other products, benzoic acid, and finally the inert base aconin.

The identification of aconitin in toxicology rests upon physiologic effects, and less clearly upon chemical reactions, as follows:

1. **Tingling of the Lip.**—When an aqueous solution of the free alkaloid or one of its salts is applied to the lip or tongue, numbness and tingling are felt, the effect passing away very slowly. A single drop may be applied. If the solution be so strong as to cause burning, it should be diluted, and care should be exercised to avoid taking an injurious dose. According to Squibb,[1] the alkaloids of $\frac{3}{10}$ of a grain (0.02 gm.) of good aconite root, in solution made up to 1 fluidram (4 c.c.), held in the anterior part of the mouth, previously rinsed with water, for sixty seconds and then ejected, give a tingling sensation, beginning in five or ten minutes and lasting about one and one-half hours, as an average effect upon different persons. When the soluble part of only $\frac{1}{10}$ of a grain (0.006 gm.) of the root was taken, the tingling effect began after fifteen minutes and lasted for from one-quarter to one-half hour.

2. **Physiologic Test Upon an Animal.**—The final solution, prepared as for test 1, is given hypodermically to a mouse, a frog, or other small animal. The death of the animal, usually within an hour, gives evidence of the poison.

3. The general reagents for alkaloids, as stated on page 452, give precipitates with aconitin. Auric chlorid gives a yellow, amorphous precipitate that can be crystallized from alcohol.

4. Moisten the residue with nitric acid, evaporate to dryness, cool, and add a drop or two of alcoholic solution of caustic potash to obtain the odor of ethyl benzoate. This test is not applicable to quantities as small as those usually in question in toxicology.

5. If poisoning by aconite as a crude drug or fresh plant be in question at all, the contents of the stomach should be inspected for fragments of roots, leaves, or bark, and these subjected to a botanical examination. A microscopic examination for the powder of the root may be made upon the collected and washed residue of the contents of the stomach.

Separation of Aconitin from the Tissues.—This is to be undertaken with precautions against hydrolysis of the alkaloid. The process elsewhere directed for atropin is to be employed, but with less exposure to heat in the evaporations, employing vacuum or a current of dry air instead of the heat of a water-bath.

Deposition in the Body.—Beside the stomach, the urine, the kidneys, and the liver are to be examined. Aconitin is excreted especially in the urine and the saliva (Kobert).

Failure to Detect.—The conclusion of Professor Haines[2] is certainly just when he says: "If a small but fatal dose of the poison

[1] E. R. Squibb, *Ephemeris*, 1882, vol. i., p. 125.
[2] Hamilton's *System of Legal Medicine*, vol. i., p. 423.

were to be given, especially if it were administered hypodermically, the chances of its detection in the body after death would not be great."

ATROPIN AND RELATED ALKALOIDS.

General Description and Relations.—Atropin is a poison found in *Atropa belladonna* (deadly nightshade). It is the best known representative of a family of poisonous alkaloids found in plants of the *Solanaceæ*. Atropin, $C_{17}H_{23}NO_3$, is related to hyoscyamin, hyoscin, and scopolamin. Hyoscyamin suffers conversion into atropin during certain chemical operations and in drying the drug.[1] Homatropin is an

FIG. 41.—Belladonna, or deadly nightshade (*Atropa belladonna*).

artificial alkaloid having its chief chemical part in common with that of atropin and giving briefer effects. The same is true of benzoyl tropein. All these alkaloids dilate the pupil of the eye to so marked an extent that they are classed as the **mydriatic alkaloids.** Their poisonous action includes delirium, various paralytic effects, suppressed secretions, sometimes convulsions; the different members of the family agree in their leading characteristics.

[1] Optically inactive atropin appears to be a racemic union of dextro-atropin and levo-atropin. The latter is found by Amenomiga to be identical with hyoscyamin. *Archiv der Pharmacie*, 1902, vol. ccxl., p. 498.

The form of atropin more commonly furnished is the sulphate,—$(C_{17}H_{23}NO_3)_2H_2SO_4$,—occurring in white crystals, freely soluble in water or alcohol. The free alkaloid is sparingly soluble in water.

Symptoms of Poisoning by Atropin and its Related Alkaloids.—Poisoning by belladonna, hyoscyamus, stramonium, and *Duboisia myoporoides* is due to the alkaloids of the atropin family, and is attended by the same leading symptoms—marked dryness of the mouth and throat, redness of the tongue, thirst, difficulty in swallowing, wide dilatation of the pupils, impaired vision and loss of all accommodation of the eye, hallucinations, then well-marked delirium, excitement, and sometimes chorea-like movements or convulsions. Nausea is common and vomiting is not unusual. The pulse becomes feeble and

Fig. 45.—Stramonium (*Datura stramonium*).

rapid, and sometimes intermittent. A deep-red eruption upon the skin has been observed in many cases. In the later stages numbness and paralysis of the limbs sometimes occur, and before death there is generally coma, and occasionally there are convulsions.

The external application of atropin, or of preparations of belladonna or hyoscyamus, is liable to produce their poisonous effects.

The dilatation of the pupil is due to local action.[1] Introduced into the circulation at large, the poison affects both eyes; applied externally to one eye, its pupil only is dilated. The pupil is dilated to some extent in some stages of the action of cocain, and sometimes under the action of digitalis and of coniin, but these effects do not simulate the

[1] Schmiedeberg, *Pharmacology*.

extent and persistent dilatation of atropin poisoning. The "ptomatropins" from badly preserved meats, etc., cause dilatation of the pupil and other symptoms like those of atropin.[1]

The poisonous effects of the several natural tropeins—hyoscyamin, hyoscin, belladonnin, scopolamin—do not show characteristic differences sufficient to enable the observer to distinguish one from the other by the symptoms. Homatropin, an artificial tropein, is much milder in its poisonous effects, and the dilatation of the pupil, although equally de-

FIG. 46.—Henbane (*Hyoscyamus niger*).

cided, appears earlier and passes away much sooner than that of the natural tropeins.

Period when Fatal.—The symptoms of poisoning by atropin and its related alkaloids appear within a few minutes after absorption of the drugs into the blood, and usually, although not invariably, within

[1] Consult p. 703 in section on Ptomains and Other Bacterial Products in Their Relation to Toxicology; also, Vaughan and Novy, *Cellular Toxins*, 1902, p. 244; Kobert, *Intoxikationen*, 1893.

an hour after the drugs or the alkaloids have been taken into the stomach. The usual course of the poisoning is not rapid, and in a probable majority of fatal cases death has occurred within twenty-four hours from the time when the poison was taken.

Fatal Quantity.—Constitutional differences are so great in the power to recover from atropin poisoning that any statement as to the smallest fatal dose is liable to be misleading. Recovery occurs in a majority of the cases. According to the statistics of Falck, quoted by Kobert,[1] recovery was the result in 88.4 per cent. of the cases collected. In this country an extraordinary case has been reported[2] in which death resulted from the hypodermic administration of $\frac{1}{30}$ of a grain (0.00216 gm.) of atropin, and there are recorded numerous cases of dangerous results from like doses in hypodermic use. In other cases, however, recovery has followed poisoning by hypodermic administration.

Treatment.—The aim is—(1) To remove the contents of the stomach and wash the organ out; (2) to improve any possibility of precipitation of any unabsorbed portion of the alkaloid; (3) to combat carefully the absorbed alkaloid by the physiologic effects of morphin, possibly of pilocarpin.

1. For the first indication use a siphon-tube and water at ordinary temperature; this is more effective than emetics.

2. If it be undertaken to precipitate any of the alkaloid in solution in the stomach before the evacuation of this organ, administer 6 minims of compound solution of iodin diluted with water (3 or 4 ounces). The weight of the free iodin should be as much as four times the weight of the atropin to be precipitated. The stomach should be washed out without delay after the iodin is given. Instead of the iodin, an excess of tannic acid may be employed (see Case 9). The administration of finely powdered charcoal is a resource, instead of either of the precipitants just named, to hold the alkaloid for a short time and retard its absorption.

3. To oppose the poison by the physiologic effects of morphin the latter is administered hypodermically in full medicinal doses, repeated as indicated by their effects upon the eye, the pulse, and the respiration (see under Morphin, p. 492, and under Atropin, Cases 8, 11, 22). Pilocarpin was administered in Cases 17 and 19; physostigmin in Cases 16 and 18; potassium bromid and chloral in Case 10.

Cases of Poisoning by the Tropein Alkaloids or by Drugs Containing Them.

Case 1.—A woman fifty-nine years of age took 1 grain (0.065 gm.) of atropin sulphate. The symptoms that followed were glassy eyes and dilated pupils, delirium, great thirst, numbness of hands and feet, the surface of the entire body being a scarlet-red, pulse 100, temperature 101° F., and frequent urination. Recovery.[3]

Case 2.—A man of thirty-six years was treated with $\frac{1}{16}$ of a grain (0.004 gm.) of atropin hypodermically. Dryness of mouth and throat, dimness of sight with dilated pupils, full, strong pulse (76), breathing quiet and regular. Recovery.[4]

[1] *Intoxikationen*, 1893, p. 606. [2] Arnold, *Baltimore Med. Jour.*, 1871, p. 169.
[3] L. Ott, *Med. News*, Philadelphia, 1895, vol. lxvii., p. 628.
[4] C. Bing, *Berlin. klin. Wochenschr.*, 1895, vol. xxxii., p. 997.

CASE 3.—A woman took 3½ grains (0.225 gm.) of atropin sulphate. Symptoms that followed consisted of widely dilated pupils, numbness, weak pulse (70), spasms, and insensibility for nine hours. Treatment consisted of emetics and brandy and water. Recovery.[1]

CASE 4.—A woman of twenty-five years took about ⅓ of a grain (0.02 gm.) of atropin sulphate. Hysteric screaming, loss of accommodation of the eyes, convulsions, very weak and slow respiration, great thirst, and profuse urination followed. Recovery.[2]

CASE 5.—A woman of twenty-six years took ¾ of a grain (0.05 gm.) of atropin sulphate, which was followed by furious delirium, dilated pupils, and collapse. Treatment consisted of morphin hypodermically. Recovery.[3]

CASE 6.—A boy of seven years was treated with *an external application* of a solution of atropin to relieve a burn. Delirium, great thirst, dilated pupils, dimness of vision, pulse 160, temperature 99° F., followed. Treatment by cognac and turpentine. Recovery.[4]

CASE 7.—A woman of forty years took ½ of a grain (0.033 gm.) of atropin sulphate. Symptoms were dilated pupils and diminished vision, weak pulse of 116, temperature 96⅗° F., respirations 3 a minute. Treatment by morphin and coffee. Recovery.[5]

CASE 8.—A child of two years took ⅓ of a grain (0.022 gm.) of atropin. Symptoms consisted of dilated and immovable pupils, dry skin, scarlet rash, a pulse of 160, and restlessness. Treatment: morphin subcutaneously. Recovery.[6]

CASE 9.—A woman took ½ of a grain (0.03 gm.) of atropin. Symptoms: dilated pupils, delirium, unconsciousness, pulse 100. Treatment: cognac, iodin in solution of potassium iodid, tannic acid. Recovery.[7]

CASE 10.—A child of three years ate stramonium seeds. The symptoms that followed consisted of dilated pupils, bright-red, hot and dry skin, full pulse, tetanic contortions. Treatment: emptying the stomach, potassium bromid, chloral hydrate. Recovery.[8]

CASE 11.—A boy of three years ate some "thorn apples" (*Datura stramonium*). Pupils became widely dilated, pulse 160, respiration 64, with occasional convulsions. Treatment: castor oil and morphin. Recovery.[9]

CASE 12.—A boy of six years ate berries of *Atropa belladonna*. Dilated pupils, delirium, unconsciousness, dryness and itching of the skin, pulse of 120 followed. Recovery.[10]

CASE 13.—Children ate of the root of *Hyoscyamus niger*. Thirst, redness and heat of skin, hallucinations, incontinence of urine, and vomiting ensued. Recovery.[11]

CASE 14.—A woman applied a belladonna plaster to her breast. Dimness of sight, thirst, and a rash upon the limbs followed.[12]

CASE 15.—A woman applied a belladonna plaster over the lower portion of the back. There followed dryness of the mouth, throat, and skin, hyperesthesia of the senses, symptoms peculiar to locomotor ataxia, and frequent and excessive urination.[13]

CASE 16.—A man of fifty-seven wore "a strong belladonna plaster" over his loins. There resulted dryness of the mouth and tongue, numbness of the hands and feet, dimness of sight, delirium, and a comatose condition. The skin under the plaster was very cold. Treatment: physostigmin. Recovery.[14]

[1] E. A. B. Traverse, *Med. Jour.*, London, 1889, vol. i., p. 1051.
[2] G. Bentzen, *Schmidt's Jahrb. d. Med.*, 1885, vol. ccviii., p. 131.
[3] Crozier, *Amer. Jour. Med. Sci.*, 1875, vol. lxix., p. 574.
[4] Kjelberg, *Schmidt's Jahrb. d. Med.*, 1881, vol. cxci., p. 129.
[5] L. Eliot, *Med. Record*, New York, 1883, vol. xxiv., p. 372.
[6] Warfvinge, *Schmidt's Jahrb. d. Med.*, 1885, vol. ccv., p. 135.
[7] F. Ockeck, *ibid.*, 1885, vol. ccviii., p. 131.
[8] J. M. Pace, *Med. and Surg. Reporter*, Philadelphia, 1881, vol. xlv., p. 26.
[9] H. Terry, *Boston Med. and Surg. Jour.*, 1882, vol. cvi., p. 123.
[10] S. R. Scofield, *Lancet*, London, 1895, vol. ii., p. 199.
[11] Pipping, *Schmidt's Jahrb. d. Med.*, 1885, vol. ccviii., p. 130.
[12] Griffith, *Brit. Med. Jour.*, 1891, vol. i., p. 1060.
[13] E. E. Maddox, *Amer. Jour. Med. Sci.*, Philadelphia, 1893, vol. cvi., p. 572.
[14] H. J. Howarth, *Lancet*, London, 1894, vol. i., p. 204.

CASE 17.—A woman of forty-two years took 2 ounces and 2 drams (64 c.c.) of belladonna liniment. The pupils became dilated, the face swollen, the pulse almost imperceptible, the respirations numbered 25, and there was a profound stupor. Treatment: pilocarpin hypodermically. Recovery.[1]

CASE 18.—A man of thirty-five years received a hypodermic injection of morphin and atropin together. He became delirious and unconscious. Treatment: physostigmin hypodermically. Recovery.[2]

CASE 19.—A woman of thirty-seven years took a tablespoonful of belladonna liniment. The pupils became widely dilated, the breathing stertorous, and the patient entirely unconscious; the heart was weak and greatly excited, the extremities were cold. Treatment: pilocarpin and coffee hypodermically. Recovery.[3]

CASE 20.—A woman of seventeen years took two tablespoonfuls of mixed liniments, *belladonna and aconite*. Symptoms: dilated pupils, pulse barely perceptible, heart-beats reaching 300, teeth clenched, violent spasms, and stertorous breathing. Death occurred in one hour and twenty minutes.[4]

CASE 21.—A man of thirty-three years took a mixture of more than half an ounce of belladonna liniment, two teaspoonfuls of laudanum, and a little camphorated chloroform. Symptoms: widely dilated pupils, staring eyes, violent rolling of the head, sore throat, and unconsciousness. Recovery followed without treatment.[5]

CASE 22.—A woman of twenty-seven years took a mixture of atropin and morphin. There was deep coma, the skin was cyanotic and cool, the pupils were greatly dilated, the respirations numbered 5 or 6, pulse 90, and there were delirium and retention of urine. The patient recovered. Some of the contents of the stomach were found to contain both atropin and morphin, and both these alkaloids were also found in the urine.[6]

CASE 23.—A man of forty-five years took at least a teaspoonful of *liquor atropiæ sulphatis* (nearly 1 per cent. strength) on August 6th, and died of the poisoning on August 10th. The poison was taken at 1 P. M., and the pupils were not affected until 1.39 P. M., becoming almost fully dilated by 2.45 P. M. Death occurred on the fourth day. At autopsy the pupils were found slightly dilated, the heart enlarged and very soft, the liver and kidneys soft, the body not rigid, the abdomen swollen and tympanitic.[7]

Postmortem Appearances.—The pupils are found dilated. The vessels of the brain are heavily charged with blood. Redness of the mucous membrane of the stomach has been reported. The blood is generally liquid and of a dark color.

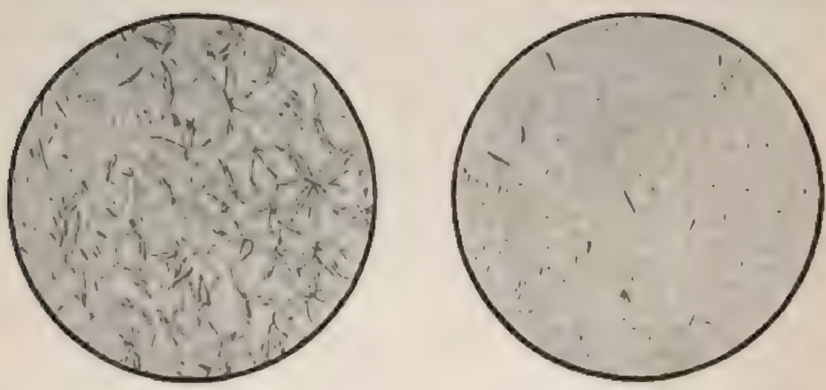
Chemical Tests.—The atropin family of alkaloids, called tropeins, have a chemical constitution now fairly well ascertained to consist of a tropin in ester-like combination with certain tropic acids. A tropin is a quite direct derivative of the pyridin type, and the tropic acids are phenylhydroxy-propionic acids. The acid of homatropin is phenyl-hydroxy-acetic acid. All the tropeins, therefore, are saponifiable into a tropin and a hydroxy acid, the tropin being a tertiary nitrogen base, and the acid containing the phenyl group. The structure of a tropin corresponds to that of ecgonin, the nucleus of cocain, but is distinctly different. Merling[8] infers that both of these bodies have a double-

[1] N. Grattan, *Lancet*, London, 1881, vol. i., p. 951.
[2] J. Hudson, *Brit. Med. and Surg. Jour.*, 1881, vol. i., p. 918.
[3] McGowan, *Lancet*, London, 1890, vol. ii., p. 175.
[4] E. H. Lipscomb, *Brit. Med. and Surg. Jour.*, 1888, vol. i., p. 694.
[5] G. H. Biden, *Brit. Med. Jour.*, 1891, vol. i., p. 284.
[6] F. Deutschmann, *Schmidt's Jahrb. d. Med.*, 1883, vol. cxxvii., p. 234.
[7] A. S. Greenway, *Brit. Med. Jour.*, 1878, vol. ii., p. 516.
[8] *Bericht d. chem. Gesellschaft*, 1891, vol. xxiv., p. 3108; see also Willstatter, *ibid.*, 1897, vol. xxx., p. 731; further, on the chemistry of all atropin alkaloids, see A. Pinner, *Chem. Centralbl.*, 1898, vol. i., p. 679.

chain structure. All the characteristics of the tropeins go to confirm the conclusions of the synthetic chemists that they are strongly individualized compounds, as indicated by their physiologic effects.

For the identification of atropin, in common with the other tropeins, there are especially—(1) The physiologic test upon the eye; (2) the color test of Vitalli; (3) the crystalline form of the bromin compound; (4) odor tests of aromatic products.

1. **The Physiologic Test Upon the Eye.**—The eye of man or of the cat or the dog is employed, and for the test there is to be prepared an aqueous solution of known strength of the sulphate, acetate, or free alkaloid, not alkaline, nor more than very slightly acidulous, nor strongly saline, and not alcoholic. One or two drops are introduced from a pipet into one of the eyes, the pupil of which is compared in size with its fellow from time to time. In the results of E. R. Squibb [1] with the eye of man, using one drop of the solution, a dilution to 2280 parts gave commencing



a *b*

FIG. 47.—Atropin treated with bromin: *a*, $\frac{1}{100}$ grain atropin in 1 grain water (× 75); *b*, $\frac{1}{1000}$ grain atropin in 1 grain water (× 125).

dilatation in from fifteen to eighteen minutes; a dilution to 4560 parts, in thirty minutes. It would appear, therefore, that $\frac{1}{2000}$ of a grain (0.00003 gm.) of the alkaloid in a drop of solution is sufficient to show dilatation in about half an hour.

2. **The Test of Vitalli.**—The dry residue from a drop or two of a quite concentrated solution on white porcelain is barely moistened by concentrated sulphuric acid at the point of a narrow glass rod, when about a milligram of solid sodium nitrite is added and rubbed into the spot without spreading it needlessly. The spot is now touched with a freshly prepared saturated solution of caustic potash in hot absolute alcohol, added in excess of the acid. Or, as in the original method of Vitalli, the spot is first touched with a drop of fuming nitric acid, evaporated to dryness on the water-bath, and then treated with a drop of the alcoholic solution of potash. The tropein reaction is a fine violet color, soon fad-

[1] *Ephemeris*, 1885, vol. ii., p. 855.

ing to dark red and disappearing.[1] (See No. 1, Plate 7.) The $\frac{1}{3000}$ grain (0.02 mg.) of the alkaloid is sufficient to give a good reaction. Whether or not the reaction of Vitalli is given by the "ptomatropins"—bodies of unknown composition found in badly preserved meats—does not appear to be settled.

3. **The Bromin Test of Wormley.**—To a drop or two of an aqueous solution of a salt of the supposed alkaloid, on a glass slide, over black paper, is added a drop of aqueous solution of hydrobromic acid saturated with free bromin. The presence of most alkaloids is indicated by an amorphous precipitate of light-yellow color. In the case of atropin or hyoscyamin the precipitate soon acquires a characteristic crystalline form, which should be compared with that from known atropin in a parallel test.[2] Hyoscin produces a precipitate of minute globules which, under certain circumstances, may change to groups of bold crystals quite different from those obtained with atropin or hyoscyamin.[3]

4. **The Test for Products of Aromatic Odor.**—If $\frac{1}{64}$ of a grain (1 mg.) of atropin be heated in a small test-tube until white vapor arises, then about one cubic centimeter of concentrated sulphuric acid be added and heated until the acid begins to color, and then, after cooling sufficiently, about two cubic centimeters of water be added drop by drop along the side of the tube, an odor suggestive of flowers will be obtained. If a small crystal of potassium permanganate be introduced before the water is added, the odor will be like that of bitter almonds.

The tropeins are precipitated from their aqueous solutions by the general reagents for alkaloids.

Atropin, free alkaloid, pure, melts at from 114° C. (Ladenburg) to 115°–115.5° C. (Schmidt). It crystallizes in needles or acicular prisms. From alcohol or from ether solution it readily forms long rectangular prisms, transparent, of the rhombic system. Is often found amorphous, also yellowish.

The Separation of Atropin, Hyoscyamin, etc., from Animal Tissues.—The material in a weighed portion is finely divided by playing upon it in a good-sized evaporating dish with a pair of large shears of bright surface. The divided material is digested with five or six volumes of diluted alcohol (about 50 per cent.), with the slightest acidulation by acetic acid, stirring constantly for half an hour. The mixture is strained, the residue digested about eight minutes with a smaller portion of the same solvent, and strained again, washing with a third and smaller portion of the solvent. The mixed alcoholic solutions are digested while stirring on the water-bath for fifteen or twenty minutes, adding a little full-strength alcohol from time to time, nearly to complete the precipitation caused by the alcohol, when the mixture is strained and the residue washed with a little alcohol of about the same strength as that of the filtrate. The liquid is now treated with sufficient calcined magnesia to take up the free acid, and concentrated by distilling rapidly

[1] D. Vitalli, 1880; Arnold, 1882; Fluckiger, *Reactions* (Nagelvoort's translation), 1893, p. 17.
[2] Wormley, *Micro-Chemistry of Poisons*, second ed., p. 642.
[3] *Ibid.*, *Amer. Jour. Pharm.*, November, 1894 p. 514.

in the vacuum obtained by a water-pump[1] as long as the liquid remains limpid. It is now strained and the residue washed with a little solvent of about the same alcoholic content as the filtrate. The latter, if too much loaded with tissue matters, so as to cause the liquid to emulsify in shaking out afterward, may be treated with full-strength alcohol, filtered—better by use of a filter-pump and a Buechner funnel—and the filtrate concentrated as before.

The extraction, however varied, should be so governed as to avoid, so far as possible, the conditions of hydrolysis.

The final aqueous liquid, which may be slightly alcoholic, filtered clear, is gently shaken in a separator with chloroform, then made only perceptibly alkaline by the addition of ammonia, again shaken (not so violently as to cause emulsification) for about five minutes, when the crude chloroform extract is drawn off, and in the same way a second and a third portion obtained. The total crude chloroform extract is now shaken out with water very slightly acidulated with sulphuric acid in three portions, and at once the total water solution is shaken with chloroform, then made just alkaline with ammonia, and shaken successively with three portions of chloroform. If, on evaporation, a small portion of the last chloroform extract shows the presence of obstructing tissue matter, the purification is repeated, beginning with the treatment by acidulated water, until a purified chloroform extract is obtained. This is to be concentrated, and then portions of a drop or two or three are separately evaporated to dryness for final tests, as specified under the head of Chemical Tests.

To estimate the quantity of atropin in the extract, its precipitation as a periodid, in the gravimetric method, is probably the most expedient way. The alkaloidal solution is *added to an excess* of decinormal aqueous iodin solution with potassium iodid, the precipitate well stirred, drained, washed, dried, and weighed as atropin hydriodid octaiodid. When this weight is multiplied by 0.202, the product expresses the quantity of atropin as free alkaloid.[2] Any estimate of the quantity of atropin in the tissues must depend upon an estimation of the loss of the alkaloid in the process of extraction, and if this be more than conjecture, it must be based upon the loss of alkaloid in a parallel analysis made for control.

Deposition in the Body.—Wormley recovered atropin from the blood after administering the drug to dogs and cats. The tropeins generally appear in the urine of persons poisoned by them. Atropin has been repeatedly recovered from putrefactive animal tissues, in which it does not readily decompose. After its administration, Dragendorff detected atropin in the urine, in the liver, and in the kidneys of the cat, but none was found in the spleen. It was found in the blood of a cat, weighing 6 pounds (2800 gm.), twenty-four hours after 2.8 grains

[1] The apparatus shown in Fig. 29, p. 336 is useful for this purpose.

[2] Prescott and Gordin, "Atropin Periodids," etc., *Jour. Amer. Chem. Soc.*, 1898, vol. xx., p. 329; *ibid.*, "Volumetric Estimation of Alkaloids," etc., vol. xx., pp. 712, 722, 724.

(0.1863 gm.) of the alkaloid had been administered by the mouth, with ligation of the esophagus. The same investigator gave 1 grain (0.06 gm.) of atropin daily to a rabbit of 3½ pounds (1500 gm.). Each day the urine was found to contain much atropin. On the tenth day the blood was tested and mere traces of the alkaloid found in it. In a similar experiment like results were obtained in the urine, but no alkaloid was found in the feces.

COCAIN, ALKALOID OF THE COCA-LEAF.

General Description.—Cocain is manufactured from the leaf of *Erythroxylon coca* of South America, and constitutes about 0.75 per cent. of the freshly dried leaf. Coca-leaf and its liquid preparations are furnished in pharmacy, but they are hardly known as a source of poisoning, and the same is true of the minor alkaloids of the leaf. Cocain, usually as the hydrochlorid, is in common use as a local anesthetic. It is a white, crystalline solid of a bitter taste, and, as a salt, is freely soluble in water or alcohol. Its chemical formula is $C_{17}H_{21}NO_4$. It is most used as the hydrochlorid of cocain, $C_{17}H_{21}NO_4HCl.2H_2O$, which is of variable crystalline or amorphous forms. Applied to the mucous membrane it causes a local insensibility to pain or injury. Crude cocain is shipped to manufacturers from South America, but is not found in the retail market.

Symptoms of Poisoning by Cocain.—Whether taken by the mouth, hypodermically, or by absorption through an abraded surface or a portion of the mucous membrane, if a poisonous quantity be taken into the system, severe symptoms quickly appear. There are excitement, fulness and pain in the head, dryness and burning in the throat and nose, and burning pain in the stomach. The pulse is quickened at first, and later becomes feeble and slow. The respirations at first usually are quickened, becoming slow and labored, with cold extremities and a bluish face. The pupils of the eyes are commonly dilated for a time. Cases are on record showing great deviations from the symptoms just described.

Chronic poisoning, with which may be classed the effects of the cocain habit, is a subject for the physician rather than for the toxicologist.

Period when Fatal.—In acute poisoning by cocain the symptoms appear very quickly after the poison is absorbed and run a rapid course. It is stated that, generally, if the patient survives half an hour, he recovers [1] (see the cases quoted below).

Fatal Quantity.—(See the Cases of poisoning given below, especially Cases 1 to 8.)

Treatment.—When the poison has been taken by the mouth, the first thing to do is to draw off the contents of the stomach by means of a siphon-tube or a stomach-pump, introducing water and drawing it off again, until the stomach is well washed out. If it can be done with-

[1] Walter S. Haines in Hamilton's *Legal Medicine*, vol. i., p. 428.

out delay, finely powdered charcoal may be administered during the evacuation of the stomach.

The symptoms, variable as they are, should be met by the restorative measures directly indicated. Carbonate of ammonium, hot alcoholic drinks, stimulants hypodermically, sinapisms over the heart and stomach, and friction of the extremities are helpful, especially in the cyanotic stage. Professor Haines[1] states that "inhalations of amyl nitrite and hypodermic injections of nitroglycerin are often signally useful; and inhalations of pure oxygen are very valuable in relieving threatened asphyxia. In case breathing ceases, artificial respiration should be resorted to, and electricity may be tried, with some chance of a favorable result."

Cases of Poisoning by Cocain.

Cases 1-3.—One dram (4 c.c.) of a 4 per cent. solution of cocain (about 2¼ grains—0.146 gm.—of the alkaloid) was thrown into the urethra of a man. Convulsions appeared at once, and death occurred in four minutes. The autopsy showed intense congestion of the lungs.

A 4 per cent. solution of cocain was freely applied to the face of a young woman who was treated for facial blemish. The patient walked to the window and fell dead.

Forty minims (2.5 c.c.) of a 4 per cent. solution of cocain (about 1½ grains—0.097 gm.—of the alkaloid) were injected into the seat of rectal fistula of a man of twenty-six years. In three minutes he became unconscious and convulsed, and one minute later died.[2]

Case 4.—Half a grain (0.032 gm.) of cocain was injected into the gum. The patient, a woman, became wildly delirious, the pupils were dilated, the extremities cold, the respirations hurried and irregular. Treatment by injections of brandy and ether into the rectum was followed by recovery.[3]

Case 5.—One grain (0.065 gm.) was injected into an external hemorrhoid which was to be removed. The pulse became indistinguishable, the heart-beats 40 a minute, the hands and arms in a strong clonic spasm, the extremities cold, the pupils widely dilated, and the urine suppressed. Patient recovered.[4]

Case 6.—Twenty drops of a 6 per cent. solution of cocain (about 1¼ grains—0.078 gm.—of the alkaloid) were given a man hypodermically. There was sudden collapse, with great dilatation of the pupils, quickened breathing, irregular pulse of 160, symptoms from which the patient recovered.[5]

Case 7.—A man took 4⅘ grains (0.312 gm.) of cocain hydrochlorid. Symptoms: failure of eyesight, loss of use of the legs—appeared like a drunken man—cramps in the abdomen, pulse from 80 to 86, profuse sweating. Recovery followed in six days.[6]

Case 7a.—A man of twenty-four years took 8 or 9 grains (over 0.5 gm.) of cocain hydrochlorid. The symptoms included numbness in the mouth, tongue, and throat, extending later to the stomach and abdomen, also muscular movements resembling those of a bad case of chorea, the movements, however, being slower and more regular. Treatment: nitrite of amyl, calomel, castor oil. Recovery.

Case 8.—A man suffering from disease of kidneys took 20 grains (1.3 gm.) of cocain and died an hour after. At the autopsy the lungs were found congested, as were the brain and the underlying part of the stomach. There was blood-clot in the heart. It was inferred that death occurred from paralysis of both the cardiac and respiratory centers.[7]

[1] Walter S. Haines in Hamilton's *Legal Medicine*, vol. i., p. 429.
[2] J. B. Mattison, *Dublin Jour. Med. Sci.*, 1895, vol. xcix., p. 116.
[3] C. E. Gooding, *Lancet*, London, 1888, vol. i., p. 394.
[4] J. Miller, *New York Med. Jour.*, 1894, vol. lx., p. 660.
[5] N. Teeter, *Therap. Gaz.*, 1895, vol. xix., p. 11.
[6] C. S. Kilhman, *Lancet*, London, 1887, vol. i., p. 17.
[7] *Ibid.*, London, 1889, vol. i., p. 292.

CASE 9.—A girl of sixteen years took about 10 grains (0.64 gm.) of cocain in 10 per cent. solution on an empty stomach and then drank hot tea. Death occurred in forty minutes.[1]

CASE 10.—A girl of seventeen years took from 12 to 15 grains (almost 1 gm.) of cocain and died in forty minutes. At the autopsy the pupils were found dilated, the heart-valves normal, with a small quantity of dark, fluid blood in the right ventricle, the left ventricle being empty, the lungs congested and highly crepitant, the brain anemic but its meninges engorged, the liver, spleen, and kidneys hyperemic, the bladder containing 3 or 4 ounces of urine.[2]

CASE 11.—A girl took a teaspoonful of cocain in a glass of beer and died in great pain half an hour after. At the autopsy the brain, pons, and medulla were found engorged, and there was red serum in the side ventricles. The liver, kidneys, and spleen were congested. The stomach contained dark-red mucus, yielding plain chemical tests for cocain.

Postmortem Appearances.—An excess of blood has been found in the brain, liver, kidneys, and also in the lungs. The blood has been found dark and fluid. These appearances, however, are not especially characteristic of death from this poison (see Cases 8, 10, 11).

Chemical Tests.—Cocain is both a methyl- and a benzoyl-ester of ecgonin, and therefore yields to hydrolysis or saponification, giving first methyl-alcohol and then benzoic acid. Ecgonin is a base corresponding to atropin. With this well-ascertained structure, however, no delicate and distinctive chemical tests have as yet been elaborated for cocain. Reliance may best be placed upon—(1) Physiologic tests; (2) the test for benzoic acid; (3) the crystalline form of the gold chlorid.

1. **Physiologic Tests.**—For these a neutral solution of a salt of the alkaloid is employed. Brought by a glass rod into contact with the tongue or lip, repeating the contact upon the same spot, cocain gives a sense of numbness and a characteristic insensibility to touch. (If not too dilute, there is a slight bitterness of taste.) With full effect there is a blanching of the color of the mucous membrane. The cessation of sensation lasts but a few minutes unless the application be repeated. A solution of 4 per cent. causes both numbness and blanching in marked degree. The strength of a solution obtained in analysis should be judged by comparison with graded solutions of known strength of cocain upon the same subject. Upon the eye the anesthetic and blanching effects upon the conjunctiva are obtained, and an additional test, that of dilatation of the pupil; this effect is not so extreme as that of atropin, and much less persistent.

2. **The Test for Benzoic Acid.**—Unless made with drop quantities and the use of a magnifier, this test must fail from lack of delicacy in ordinary analysis for poisons left in the body after death. To a well-concentrated, clear, aqueous solution—which need not exceed one drop—on a glass slide, add about one-tenth its volume of concentrated sulphuric acid, and heat the liquid on the water-bath for about five minutes. Add concentrated ammonia from a capillary pipet to nearly neutralize, making sure, by a minute slip of litmus-paper, that the reaction is not alkaline. Now add from the capillary tube a very little

[1] G. M. Johnston, *Brit. Med. Jour.*, 1895, vol. ii., p. 1162.
[2] O. H. Garland, *Lancet*, London, 1895, vol. ii., p. 1104.

ferric chlorid solution. A buff-colored precipitate should be obtained if cocain was present in sufficient quantity in the liquid taken. Ferric benzoate is not soluble in very dilute acetic acid. The slide may be placed alternately over white and black ground, under the magnifier, in judging of the result. A control test should be made.

3. **The Gold Chlorid Test.**—A solution of cocain hydrochlorid, which may be prepared by careful treatment of a film of residue of the free alkaloid with a drop or two of very dilute hydrochloric acid, is at once precipitated by auric chlorid when the alkaloidal solution is as strong as 1 : 3000. Under the microscope stellate crystals are discerned. In solutions of 1 : 12,000 the crystals are obtained. The test should be conducted on the scale indicated in the preceding paragraph, and if the precipitate be amorphous, it should be warmed, and then, if necessary, diluted, to obtain the crystals upon gradual cooling. Free cocain crystallizes in monoclinic prisms melting at from 97° to 98° C.

FIG. 48.—Cocain with gold chlorid.

Separation from Animal Tissues.—The method of extraction directed for atropin (p. 469) is suitable for cocain. Instead of chloroform as an immiscible solvent, benzene may be employed.

According to Sonnie-Moret,[1] cocain is somewhat quickly changed in the living organism, and unless taken in considerable quantities, toxicologic examination will give negative results.

COLCHICUM AND ITS ALKALOID, COLCHICIN.

Colchicum autumnale, the meadow-saffron, owes its poisonous effects chiefly to the alkaloid colchicin, the distinctive chemical product of the plant. Both the seed and the corm (root) are used in medicine and are liable to act as poison. From 0.2 to 0.4 per cent. of the alkaloid has been found in the seed; from 0.08 to 0.2 per cent. in the root; from 0.01 to 0.02 per cent. in the fresh flowers, and 0.003 per cent. in the fresh leaves. The drug is an importation in the United States from plants collected in the temperate regions of Europe and northern Africa. It was known to the ancients as a poison and as a remedy. The tincture of the seed, the fluid extract of the root and of the seed, and the wine of the root and of the seed are pharmacopeial preparations in medicinal use. There is also a solid extract directed to be made from

[1] Inaug. Diss., *Jahresb. d. Pharm.*, vol. xxvii., p. 390; *Chem. Centralbl.*, 1893, vol. i., p. 859.

the root. The alkaloid by itself is not pharmacopeial, either free or as a salt, and is very seldom used in medicinal practice.

Symptoms of Poisoning by Colchicum.—The symptoms resemble those of an irritant, with purging, vomiting, and depression. The particular responses of the body to the irritative attack show wide variation in different cases. Among the symptoms in the majority of the cases are a burning sensation in the throat, nausea, vomiting, severe colicky pains, great thirst, frequent purging, various derangements of the urinary excretion, coldness and moisture of the skin, and prostra-

FIG. 49.—Meadow-saffron or colchicum (*Colchicum autumnale*).

tion of strength. The mind is not directly affected. The irritation of the mucous membrane is a physiologic rather than a local effect of the poison, so that it results from hypodermic injection. The somewhat gradual development of the poisoning is explained by the statement that the alkaloid is not poisonous until, by oxidation, it is resolved into oxydicolchicin (C. Jacobi).

The symptoms usually come on within from one to three hours after the poison has been taken. In the majority of fatal cases from a single

dose death has taken place in about twenty-four hours. It has been delayed for several days. The excretion of this poison is very slow, so that in repeated doses it may be cumulative (Kobert).

Fatal Quantity.—Of the pure alkaloid, colchicin, $\frac{1}{3}$ of a grain (0.02 gm.) is liable to cause death. By analogy with fatal effect on dogs 1 grain (0.065 gm.) would be fatal for a man (Kobert). One-twelfth of a grain (0.005 gm.) has caused violent diarrhea. The fatal quantity of colchicum or its preparations may be estimated from these data, with recognition of the wide variation in strength of the drug, as already stated.

Treatment.—The stomach should be washed out as soon as possible; the mucous irritation may be allayed by mucilaginous drinks and opiates; the abdominal pains relieved by hot fomentations. It is not probable that an appreciable extent of saponification of the alkaloid (as referred to below) can be accomplished by an antidote in the body.

Cases of Poisoning by Colchicum.

Cases of poisoning by colchicum have been numerous, including criminal as well as accidental cases. In 1862 in England Catharine Wilson was tried for the murder of four persons, with the inference that colchicum seeds in wine or brandy had been employed. In 1894 H. N. Moyer[1] reported the case of a woman of twenty-two years who took a poisonous quantity of colchicum. There were severe chills, purging, abdominal pains, and vomiting. Death ensued in thirty-six hours. The stomach was found enlarged, the kidneys congested, the brain and heart hyperemic. Analysis of the contents of the stomach revealed no colchicin.

Grazing animals are frequently poisoned by colchicum in Europe.

Case 1.—Wine of colchicum of the strength of 4 ounces (113 gm.) of the seeds to a pint (576 c.c.) was taken by mistake by seventeen persons, each taking from 3 to 11 ounces (89 to 325.2 c.c.). The persons were from twelve to forty-five years of age. The symptoms of poisoning began in from forty-five to ninety minutes, and consisted of vomiting and bilious discharges. The purging was delayed in the cases of those who took the smaller quantities. There were severe cramps, irritation in the throat, hoarseness, intense thirst, small pulse, and slight dilatation of the pupils. Consciousness was retained, as was the muscular strength, with sleeplessness. Seven of the patients died in from nineteen to twenty-nine hours after the poison was taken, and ten recovered. Of the latter, one had taken eleven ounces (325.2 c.c.) of the wine.[2]

Case 2.—Five patients in a hospital in Toulon, France, were each given by mistake 2 ounces (57 c.c.) of wine of colchicum. The earliest symptoms appeared in about two hours. There were burning pains in the stomach, nausea and continued vomiting, abundant purging, a pale and cold surface, a small pulse, great thirst, and somewhat later than the first symptoms a sense of burning in the throat. There was tenesmus, both rectal and vesical. There was no loss of motor, sensory, or mental power. The five cases were all fatal in from nineteen to twenty-six hours. In the *postmortem appearances* there was no trace of inflammation in the throat or esophagus, the mucous surface of the stomach and intestines was softened and red, the stomach was distended with liquid, and the liver and spleen were congested. No colchicin was found in the vomited matters.[3]

Case 3.—A woman fifty-six years of age took 1 ounce (30 c.c.) of the wine of colchicum in divided doses in the course of twelve hours. There were nausea, vomiting, slight purging, heat and pain in the throat, pain in the stomach, thirst, a cold moist skin, and a feeble pulse. The symptoms continued for three days, when the patient recovered.[4]

[1] *Med. News*, vol. lxiv., p. 457.
[2] Major, *Med. Times and Gaz.*, London, March 7, 1874, p. 275.
[3] M. Roux, *L'Union Méd.*, March 27, 1855; *Lancet*, London, May 5, 1855, p. 474.
[4] *Amer. Jour. Med. Sci.*, January, 1857.

Chemical Constitution and Tests.—Colchicin is the methyl-ester of a nitrogenous base, or amid, colchicein; in other words, it is a saponifiable alkaloid. Both the colchicin and the colchicein exist in the plant. Colchicein, the product of saponification, is stated to be non-poisonous.[1] The saponification is effected with quantitative completeness by boiling two hours with $\frac{1}{40}$ normal potash.[2] The constitution of the colchicein is $C_{17}H_{13}NO.(OCH_3)_3.COOH$; of colchicin, $C_{17}H_{13}NO.(OCH_3)_3.COOCH_3$. The nitrogen is probably contained in an acid amid group, and the alkaloid *not* a pyridin derivative. The condensed formulæ are: for colchicein, $C_{21}H_{23}NO_6$; for colchicin, $C_{22}H_{25}NO_6$. Colchicinic acid has the formula $C_{15}H_9(OH)_3.NH_2.COOH$.

According to C. Jacobi,[3] colchicin oxidizes in the blood to oxydicolchicin $(C_{22}H_{25}NO_6)_2O$, and it is only as the latter compound that it exerts its poisonous effect.

Nitric acid (specific gravity, 1.40) with colchicin gives a violet-red color, gradually changing to yellow. On diluting with water and adding caustic soda solution, a fine red-yellow color is obtained. Concentrated sulphuric acid containing a trace of nitric acid dissolves colchicin with a play of colors—green-yellow, green, blue-green, wine-red, and finally yellow. Ferric chlorid added to an alcoholic solution of colchicin gives a garnet-red color.

In the above color-tests the same results are obtained with colchicin, colchicein, and oxydicolchicin.

Colchicin is precipitated from acid solutions by potassium mercuric iodid and phosphomolybdic acid. From neutral solutions by cadmium iodid and by nitric acid. In solution feebly acidulous with hydrochloric acid gold chlorid causes a yellowish precipitate crystallizing slowly.

Colchicin is slowly soluble in water and more freely in cold than in hot water. In alcohol and in chloroform colchicin is soluble in all proportions. It is not appreciably soluble in ether that is free from alcohol and from water. According to Dragendorff, colchicein is dissolved from acid solutions by benzene. Both colchicin and oxydicolchicin are dissolved from acid as well as from alkaline solutions by chloroform and by amyl-alcohol. Chloroform combines with colchicin when the chloroformic solution is carefully evaporated, the compound, $C_{22}H_{25}NO_6.2CHCl_3$, crystallizing in yellow needles. Treating these with boiling water, the colchicin separates quite pure (Zeisel).[4]

Colchicin is excreted through the bowels and in part, as colchicein, through the kidneys. The urine, the kidneys, and the intestines are most likely to yield the poison in postmortem analysis. The alkaloid is with difficulty destroyed by putrefactive matter (Obolonski). The procedure should have respect to the properties of colchicein as above stated. Extraction with chloroform from alkaline solution is preferred.[5]

[1] Paschkis, 1883, 1888; Kobert, *Intoxikationen*, p. 652.
[2] Zeisel, 1888; Gordin and Prescott, *Proceedings Amer. Pharm. Assoc.*, 1900, vol. xlviii., p. 135.
[3] *Arch. exp. Path. u. Pharm.*, 1890, vol. xxvii., p. 119.
[4] For the estimation of colchicin by saponification in an alkalimetric method, as well as its separation from the drug, see Gordin and Prescott, *Proceedings Amer. Pharm. Assoc.*, 1900, p. 133.
[5] Hilger, 1897.

Ptomains with colchicin-like reactions have been found in a body, twenty-two months after death, by Baumer,[1] and in a cadaver by Liebermann in 1885. Barillot[2] uses treatment to distinguish from ptomains as follows: The suspected alkaloid, as residue of free base, is heated with 1 c.c. (16 min.) of concentrated sulphuric acid, and 0.25 gm. (4 grains) of oxalic acid for an hour at 120° C., when the product is diluted with water. Excess of alcoholic soda is now added, followed by excess of acetic acid, and the solution extracted with chloroform. If colchicin were present to begin with, the chloroform extract would yield a yellow coloring-matter which, when dried, yields a violet-red coloration with concentrated nitric acid, and a raspberry-red with concentrated sulphuric acid. Ptomains give no reaction.[3]

CONIIN AND POISON HEMLOCK.

Conium maculatum, "poison hemlock," is a plant naturalized from Europe, growing wild in various parts of the United States. The leaves have been mistaken for parsley. The plant owes its poisonous power wholly to coniin ($C_8H_{17}N$), a volatile alkaloid. In the plant it exists in proportions averaging 1 per cent. in combination with organic acids. As a free base it is an oily liquid, strongly alkaline, vaporizing slightly from open vessels, of a pervading characteristic odor described as that of mice, the liquid boiling at 164° C. and distilling with little decomposition. The plant has been the cause of accidental poisoning, and there are recorded cases of criminal poisoning with the alkaloid, a single drop of which is probably enough to act fatally in some cases.

Symptoms and Effects.—Muscular weakness, great lassitude, faintness, pain in the head, failure of use of the extremities, dilatation of the pupils, with little disturbance of the intellect, and death by failure of the respiration. The concentrated free alkaloid is a local caustic. The poisonous effect is very rapid, and fatal results are reached in from one to three hours. Kobert states that about 2 grains (0.13 gm.) of the alkaloid is the lowest fatal dose, and that from ½ to 1 grain (0.03–0.06 gm.) is a seriously poisonous quantity. The effect is exerted upon the motor nerve-centers.

Treatment.—Evacuation and washing of the stomach, and then stimulating agents and measures, with every effort to maintain respiration, constitute the treatment.

Postmortem Appearances.—The blood is dark and fluid, the lungs are congested, as is, to some extent, the brain.

Chemical Tests for Coniin.—1. *The Odor.*—Obtained in full from the free base, less markedly from its salts.

2. The *vapor* of coniin with that of hydrochloric acid produces a white cloud in the air. If a watch-glass is moistened with free coniin, another watch-glass moistened with hydrochloric acid, and the latter

[1] *Arch. d. Pharm.*, 1887, ser. 3, vol. xxv., p. 911.

[2] *Bull. Soc. Chim.*, 1894, ser. 3, vol. xi., p. 514.

[3] For further information concerning colchicin-like ptomains, consult p. 705 *et seq.*, in section on Ptomains and Other Bacterial Products in their Relation to Toxicology.

glass fitted over the former, the upper glass will receive a deposit of white crystalline needles, the hydrochlorid of the alkaloid.

3. If coniin be touched with alloxan, a deep purple-red color is slowly developed, along with white, needle-shaped crystals. These, treated with caustic potash, give a purple color and mousy odor.

4. When coniin is warmed with sulphuric acid and dichromate of

FIG. 50.—Hemlock (*Conium maculatum*).

potassium, butyric acid is formed, and, if in sufficient amount, recognized by its odor.

5. The physiologic test may be applied upon a small animal. The reactions of coniin should be compared with those of nicotin, from which it is to be distinguished with care. Coniin is also to be distinguished from certain ptomains.[1]

In chemical constitution coniin is a propylpiperidin. It is moderately soluble in water as a free base. Nearly all the immiscible solvents dissolve it. The hydrochlorid is not soluble in ether. This salt melts at 218° C. and loses no weight at 90° C.

[1] See Vaughan and Novy, *Cellular Toxins*, 1902, p. 237.

For the separation from foods or tissues the process given under Nicotin is to be employed. This alkaloid, however, is of an order of bases so far approaching ptomains and leukomains in constitution that it is not surprising that toxicologists have found it difficult to distinguish coniin from putrefactive products.[1]

Cases of Poisoning by Coniin and by Conium Maculatum.

Case 1.—A man of sixty-five years having suffered for two years from a nervous affection of the facial muscles was directed by his physicians to take 50 minims (3 c. c.) of fluid extract of conium every half hour until he felt the effects, and then to discontinue the medication. The symptoms were recorded as they occurred by the patient's wife at his dictation. This had been his custom in taking medicines prescribed by physicians, and at times he had been his own physician. The record so obtained is as follows: "At 4.10 P. M. took 50 minims Squibb's fluid extract of conium. At 4.40 P. M. effect very decided in dizziness, relaxation of muscles and limbs. Fifty minims more then taken. Difficulty of walking immediately, and want of power to control movements. Forced to lie down, but no mitigation of spasms. Limbs and legs weak. Unable to hold head. Speech thickening some. Pain and heaviness in top and back part of head. Pulse 56. At 5.15 P. M. took 50 drops. Some nausea. Some tremor at the base of the clavicle and in muscles across the chest, just above the sternum. No diminution of spasms about the eyes nor of photophobia. At 5.25 P. M. drowsiness, inclined to sleep. At 5.40 P. M. eyes difficult to open, speech difficult, fulness in throat, prostration nearly complete. Diplopia vastly increased. At 6.10 P. M. nausea, twitchings on right side, trouble to articulate, eyes closed, fulness almost to suffocation in throat. Pulse about 60. In past six ——." At this point the patient stopped dictating. He was then seized with nausea, but was unable to vomit. He then wanted electricity applied, but while trying to apply it himself he fell back dead. An autopsy was made about sixty hours after death. In the brain there was venous congestion. The lungs were full of blood. A foreign growth in the brain was found, also certain degenerations of the arteries. Chemical analysis was not made.[2]

Case 2.—A man ate a large quantity of hemlock, believing it to be parsley. He soon afterward lost the power of walking, staggered, and finally fell, but still retained his consciousness and intelligence. In about two hours after taking the poison there was complete paralysis of the upper and lower extremities, with occasional spasmodic movements of the left leg, and the patient had lost the power of sight, deglutition, and speech, but was still sensible. His pulse and breathing were natural. The pupils became fixed, the action of the heart very feeble, and death ensued from paralysis of the muscles of respiration in three and a half hours after the poison had been taken. After death the lungs were found engorged, the heart flabby, the stomach congested, the blood fluid and dark. In the stomach were found the green leaves of the plant in the state of a pulp. Bruised in a mortar with addition of caustic potash, the characteristic mousy odor of coniin was obtained.[3]

Case 3.—*Effect of Coniin upon an Animal.*—A single drop of coniin was placed upon the tongue of a large and healthy cat. In a few seconds the animal was inclined to stand still, and manifested an unsteady gait when disturbed; in two minutes and a half it fell on its right side, then voided urine, had violent convulsive movements of the limbs, and a tremulous motion of all parts of the body. It was dead in three minutes after the poison had been administered. In another experiment the animal, being immediately placed upon its feet, stood perfectly still, and the pupils of the eyes became dilated and insensible; in forty-five seconds the legs of the animal became powerless and it sank upon its abdomen,

[1] Consult also p. 701 in the section on Ptomains and Other Bacterial Products in their Relation to Toxicology.

[2] *New York Times*, April 5, 6, and 13, 1875; Wharton and Stillé's *Medical Jurisprudence*, vol. ii., 1884, p. 613.

[3] J. H. Bennett, *Edin. Med. and Surg. Jour.*, July, 1845, p. 169.

then passed urine, had violent spasms of the extremities, and died in four minutes after the exhibition of the poison.[1]

CASE 4.—A soldier having eaten some soup containing hemlock leaves soon fell asleep; in an hour and a half afterward he was insensible and breathed with difficulty; his pulse was slow and hard; the extremities cold; the face bluish and distended with blood, like that of a person strangulated. An emetic of tartarized antimony was then administered, but it only produced vain efforts to vomit. He complained of being cold, and soon lost the power of speech and consciousness. He died in about three hours after taking the poison.[2]

CASE 5.—A man twenty-two years of age smelled of coniin contained in a bottle, inhaling deeply. He suffered weakness of the limbs, there were rolling of the eyes, headache, profuse perspiration, and a free flow of the lacrimal fluid before he recovered.[3]

GELSEMIUM AND ITS ALKALOIDS.

The rhizome and root of *Gelsemium sempervirens*, the yellow jasmine, a beautiful and fragrant climbing vine of southern United States, are pharmacopeial in medicine and actively poisonous. The poisonous alkaloid gelsemin was found in the root by Professor T. G. Wormley in 1870, who has estimated its quantity at 0.2 per cent. An allied alkaloid, gelseminin, was discovered by F. A. Thompson, of Detroit, in 1887. The distinct poisonous action of each of these alkaloids was reported in 1893 by A. R. Cushny, then in Germany, now in Ann Arbor. Gelsemium also contains a non-nitrogenous compound, highly fluorescent in its solutions, slightly acidulous, and

FIG. 51.—*Gelsemium sempervirens.*

FIG. 52.—Rhizome of *Gelsemium sempervirens.*

recognizable in small quantities by analysis. This fluorescent body, provisionally termed gelsemic acid by Wormley, is identified by E. Schmidt as beta-methyl-aesculetin, and found in other plants.

Preparations of the green or fresh rhizome are of greater medicinal value.[4] The asserted presence of a poisonous resin in the root has been denied on good grounds.[5] "Persons are reported to have been poisoned by eating honey gathered by the bees from gelsemium flowers."[6] "Specific gelsemium," in use by eclectic physicians and others, is pre-

[1] Wormley, *Micro-Chemistry of Poisons*, 1867, p. 446.
[2] Hanf in Orfila's *Toxicology*.
[3] H. Schultz, *Schmidt's Jahrb. d. Med.*, 1887, vol. ccxv., p. 143.
[4] *American Dispensatory*, 1900. [5] *Ibid.* [6] *Ibid.*

pared from the green root, preserved in transportation to the laboratory by adding ten gallons of alcohol to the barrel of cut roots (J. U. Lloyd).

The alkaloid "gelseminin" is a very rare article in medicinal use, and not pharmacopœial. A dry extract known as "gelsemin resinoid," of variable potency, has been in use, but it is discountenanced.

Symptoms of Gelsemium Poisoning.—Disturbance and relaxation of the muscles of the eye, double vision, dropping of the lower jaw, great general prostration and muscular relaxation, slow pulse, low temperature, respiration at first quickened and then slow and shallow. The heart action seems to depend on the respiration. The pupils are dilated in this general effect or by local application. The mind is not affected, unless as a result of failing respiration. The mode of death is by respiratory failure.

Professor Cushny found that the alkaloid gelsemin caused tetanic convulsions in frogs, but had no effect on rabbits, and that the alkaloid gelseminin caused respiratory failure both in frogs and in rabbits.[1]

Fatal Quantity.—It is recorded that 12 minims (0.8 c.c.) of the fluid extract proved fatal to a child three years old; three teaspoonfuls of the fluid extract caused the death of a woman; and 15 grains (0.97 gm.) of the "resinoid" above referred to caused death in an adult. Of the alkaloid gelseminin $\frac{1}{8}$ of a grain (0.008 gm.) given hypodermically to a rabbit caused death in an hour and a half (see cases below). Professor Wormley found that 8 fluidounces (236 c.c.) of the fluid extract yielded 3.2 grains (0.2 gm.) of alkaloid.

Period when Fatal.—Death usually occurs in from one to eight hours. In one recorded case death occurred within an hour. The symptoms begin almost immediately after absorption into the circulation.

Treatment of Gelsemium Poisoning.—The stomach is to be evacuated and repeatedly washed out with warm water. The warmth of the body and the circulation of the blood are to be promoted by internal and external stimulation, such as hot drinks, friction, external heat. The respiration should be stimulated in every way, including the efforts of the patient in the earlier period and artificial movements later on, to carry the patient over the brief duration of gelsemium poisoning. Digitalis has been recommended in hypodermic administration to strengthen the heart, also the joint use of morphin and atropin to strengthen the respiration, but there is not enough known of the effect of any of the narcotics to warrant their use further than to give relief under close observation.

Cases of Poisoning by Gelsemium.—Professor T. C. Wormley, in his record of later research upon this drug,[2] states: "Of 25 cases of gelsemium poisoning that we have collected (some private), 13 proved fatal. The fatal period varied from one hour to about eight hours. Very small quantities of the drug may cause death. . . . Four doses each of 15 minims of the fluid extract, repeated at short intervals, caused the death of a healthy man in less than four hours after

[1] *Berichte d. chem. Gesellschaft*, 1893, vol. xxvi., pp. 1725–1727.
[2] *Amer. Jour. Pharm.*, 1882, vol. liv., pp. 337–344.

the last dose was taken. Also a teaspoonful of the same preparation proved fatal to a woman."

Postmortem Appearances.—For the most part these, as so far found, are normal, except superficial venous injection and congestion of some of the organs.

Tests for Gelsemium Constituents.—The alkaloid gelsemin is colorless, amorphous, or obscurely crystalline, melting at 120° C.,[1] very bitter, and without odor. It is slightly soluble in water, with alkaline reaction, freely soluble in alcohol, ether, and chloroform. The two alkaloids are separable, when in sufficient quantity, by crystallizing their hydrochlorids from alcohol. The gelsemin hydrochlorid crystallizes out almost completely, leaving the gelseminin hydrochlorid in the solution. The chemical formulæ of these alkaloids have been reported upon by several chemists,[2] but agreement as to composition has not been reached as yet.[3] The difficulty in this respect has approached that met with in the aconite alkaloids, while vastly more labor has been devoted to the latter. The marked distinctness of the chemical tests of identification of gelsemium constituents, however, is in contrast with the lack of such distinctness on the part of aconite alkaloids. In the chemical tests for identification of gelsemin and gelseminin these alkaloids do not interfere with each other. In the more conclusive physiologic tests also the two alkaloids can be taken together; for full discrimination as to physiologic effect the alkaloids must be taken separately, as already indicated.

Chemical Tests for the Alkaloids.—If a colorless or very nearly colorless residue consisting of gelsemium alkaloids, on white porcelain, be touched with concentrated sulphuric acid, the residue dissolves with a slight yellowish to brownish color. (With separate gelsemin but slight color is obtained; with separate gelseminin, a slight yellowish to pale brownish color.) If now a minute solid fragment of potassium dichromate be added, a red to purple color is developed in "reddish-purple streaks along the path of the crystal."[4] The color finally becomes blue-green. (Separate gelseminin, in this test, gives very nearly the same color obtained by gelsemin or by both the alkaloids together.) If cerosoceric oxid be used instead of the dichromate, the color bears more upon bright red (see Plate 7, No. 3). "In a parallel experiment carried on with strychnin the two alkaloids cannot be mistaken."[5]

The reported color reactions with nitric acid appear to have been subject to the effects of impurities in the alkaloid residue examined. Gerrard found no reaction by fully purified gelsemin with nitric acid in the cold.

Gold chlorid gives a yellow precipitate, crystalline, soluble when heated, of the formula $(C_{12}H_{14}NO_2)_2HCl(AuCl_3)_2$, or containing 37.4 per cent. of gold.[6]

[1] Spiegel, 1893. [2] Beilstein, *Organ. Chem.*, third ed., 1897, vol. iii., p. 884.
[3] Compare A. W. Gerrard, *Amer. Jour. Pharm.*, 1883, vol. lv., pp. 256–260.
[4] Wormley, *Amer. Jour. Pharm.*, 1882, p. 342.
[5] A. W. Gerrard, *ibid.*, 1883, p. 258. [6] A. W. Gerrard, *loc. cit.*, 1883.

Physiologic Tests for the Alkaloids.—Gelsemium alkaloids, $\frac{1}{8}$ of a grain (0.008 gm.) administered hypodermically to a cat, caused marked symptoms in fifteen minutes and death in one hour and a half.[1] One-sixth of a grain (0.01 gm.) given to a frog produced, after half an hour, great prostration, followed by tetanic convulsions and death in about four hours.[2]

Cushny[3] found gelseminin to be intensely poisonous to both frogs and rabbits, causing respiratory failure in both, and also that it dilates the pupil. He found gelsemin to cause tetanic convulsions in frogs, with final deadening of sensory nerve-endings, and to have no effect on rabbits.

Chemical Tests for the Fluorescent Substance.—This is extracted by shaking out with ether from acidulous solution and evaporating off the solvent. The fluorescent body is beta-methyl-aesculetin, $C_{10}H_8O_4$, its constitution being that of a methyl derivative of aesculetin, the latter a dihydroxyl derivative of cumarin. Aesculetin is a saponification product of aesculin, a bitter principle of horse-chestnut bark. In 1876 Sonnenschein reported that the fluorescent body found by Wormley in gelsemium was aesculin, but this was shown to be erroneous, not only by Wormley,[4] but by several other chemists. In 1898 Professor E. Schmidt, of Marburg, showed the body in question to have the composition beta-methyl-aesculetin, which also exists in *Scopolia japonica* and other solanaceous plants.

The fluorescent body of gelsemium, obtained as stated above, exhibits its distinctive optical power when dissolved in caustic alkali solution. The fluorescence is greenish-blue by reflected light. The color by transmitted light is yellow. The fluorescence is distinct in a solution diluted to 100,000 times the quantity of the constituent in question.[5] The fluorescence is exhibited by the fluid extract and other aqueous solutions of the drug gelsemium on adding alkali.

The fluorescent body is a reducing agent, deoxidizing Fehling's solution when boiled, and separating silver from its ammonionitrate solution.

Physiologic Test for the Fluorescent Body.—When $\frac{1}{6}$ of a grain (0.01 gm.) of this body was administered hypodermically to a frog, in a few minutes the eyes were fluorescent and the animal sluggish. An injection of $\frac{1}{2}$ of a grain (0.03 gm.) into the peritoneum of the frog was fatal, the animal becoming apparently lifeless in five minutes and the heart ceasing to beat after forty minutes.[6]

Taken together, the available evidences of death by gelsemium poisoning, when fully obtained, are very conclusive.[7]

Separation from Tissues and Organs.—The process given under Atropin may be followed, using either ether or chloroform, and saving the ethereal extract from acidulous solution to be examined for the fluorescent body.

[1] Wormley, 1882. [2] *Ibid.* [3] *Loc. cit.*, 1893.
[4] *Amer. Jour. Pharm.*, vol. liv., p. 338. [5] Wormley, 1882 [6] *Ibid.*
[7] E. Schwartz, *Pharm. Jour. Trans.*, 1882, ser. 3, vol. xiii., pp. 148–152.

Professor Wormley was able to recover from the body and identify both the alkaloid and the fluorescent substance four and a half months after death. In this case three teaspoonfuls of the fluid extract had been taken.

MORPHIN AND OPIUM.

General Description of Morphin and of Opium.—Morphin is the most abundant of the alkaloids in opium, and the constituent to which the poisonous effects of opium are almost wholly due. As a free

FIG. 53.—Poppy (*Papaver somniferum*).

base, crystallized morphin has the chemical composition $C_{17}H_{19}NO_3.H_2O$. In opium it exists as a meconate or a sulphate or as both. The sulphate, $(C_{17}H_{19}NO_3)_2H_2SO_4.5H_2O$, is the salt of the alkaloid chiefly used in this country, but the hydrochlorate, the acetate, and the free alkaloid are among the medicines of our pharmacopeia, and many other salts are known.

Morphin sulphate is sold in white, feathery needles, of silky luster, odorless, and of an immediate bitter taste. It is freely soluble in

water, moderately soluble in alcohol, and its solutions are neutral to litmus. The free alkaloid morphin appears in a white powder or in colorless shining crystals, gradually giving a bitter taste. It is not perceptibly soluble in cold water, and it is only moderately soluble in alcohol. The morphin salts of powdered opium are freely dissolved out by water or alcohol.

Opium itself is obtained in irregular masses, more or less moist and plastic, chestnut-brown or darker. It is also obtained in powder, nearly dry, and of lighter color than the original concrete mass. Powdered opium, according to the pharmacopeia, should be of four-tenths greater morphin strength than crude opium, the latter containing moisture. Opium has "a sharp narcotic odor and a peculiar bitter taste." The pharmacopeia of the United States (1890) prescribes that crude opium shall contain not less than 9 per cent. of morphin, and "powdered opium" not less than 13 nor more than 15 per cent. of morphin. These standards of strength agree substantially with the lower limit of strength maintained by the United States Customs' Service for the importation of the article. (For defining descriptions of extract of opium, deodorized opium, pills of opium, tincture of opium, wine of opium, vinegar of opium, and other official opium preparations, see the United States Pharmacopeia.) These preparations are properly made from powdered opium, and are governed by its limits of morphin strength. This strength can be determined in any preparation by an application or modification of the pharmacopeial methods of assay. In a case of poisoning a portion of the article used should, if possible, be secured for an analysis for morphin and its quantity, and for meconic acid if the presence of opium be in question. (Further as to these analyses, see under Analytic Tests.)

In all, at least seventeen alkaloids have been found with certainty in opium,[1] but not nearly all these are present in every variety of the drug.[2] Of the alkaloids other than morphin, the greater number are either harmless or but slightly poisonous, and are present in very small quantities. Thebain is the only one of the opium alkaloids which is more poisonous than an equal quantity of morphin, although entirely different in effect, and its quantity is very small, having been reported as varying from 0.15 to 1 per cent., the higher limit being rare, if not doubtful.

Counting morphin as the only narcotic principle of opium, the United States Pharmacopeial powdered opium, which is required to have a mean morphin strength of 14 per cent., should be borne in doses 7 times as large as those of the alkaloid morphin, or 5.6 times as large as those of crystallized sulphate of morphin. The Continental pharmacopeias, as shown on page 490, make the maximum medicinal

[1] Twenty alkaloids are included in a list of those of probable identity.

[2] The most of these alkaloids originate in the exuded milky juice of the poppy capsule while the juice is drying, and through a kind of fermentation. According to Clautrian, only morphin and narcotin are preformed within the plant (Kobert, *Intoxikationen*, p. 551).

dose of opium 5 times larger, the British 6 times larger, than the corresponding dose of morphin salts.

Symptoms of Poisoning by Morphin or Opium.—In cases of acute poisoning by opium or by morphin alone taken by the mouth, the effects begin to appear, in a probable majority of cases, in from twenty to forty minutes. Taken hypodermically, the symptoms appear earlier, and narcotism sooner follows the initial symptoms, the same being measurably true under conditions of very rapid absorption from the stomach. Quantities much larger than just enough to be fatal usually cause a preponderance of comatose symptoms, sometimes from the first. On the other hand, a poisonous quantity not more than enough to be fatal gives rise to the more pronounced and prolonged initial stage—that of mental exaltation and increased action of the heart. Still wider deviations of symptoms and the time of their accession result from the individuality of the nervous system (Cases 16 to 19). Persons differ from one another in the response of the nervous centers to this poison as they differ in reaction against other attacks.

The train of symptoms is made up as follows: A sense of mental exhilaration and physical ease, with both a quickening and a strengthening of the pulse—the initial stage, especially variable, as just stated. Then follow dizziness and heaviness of head, nausea, languor, and drowsiness, the pulse being reduced in frequency some time before it is reduced in force. Nausea comes early in some cases, and if there is vomiting as an effect of the poison, the earlier it occurs the more thorough it is likely to be. In some cases there are itching of the skin and even a sudden eruption. The desire to sleep increases and returns irresistibly in spite of efforts to keep awake. With approaching stupefaction there is gradual loss of muscular power and a diminished sense of feeling. Meantime the pupils of the eyes have become contracted—a distinctive symptom. The sensitiveness of the conjunctiva is diminished, and the pupils fail to respond to light. The respirations have become less and less frequent, and are finally reduced in some cases to three or four a minute, with stertorous breathing. Shallow respiration is often observed. A peculiar respiratory pause or interval is found in some cases, lasting one-half a minute or even longer, followed by somewhat rapid breathing, becoming then gradually slower until, after twenty or thirty respirations, there is another pause. Cases have been found in which the pupil would contract at the time of the pause and dilate during the rapid breathing, and the stupor has been found to vary with the same alternation. In other cases the respiration is calm, being gradually reduced from first to last. The breathing gives an index of the action of the poison upon the respiratory nerve-center, the one point where death is threatened and defense is made. With slow or interrupted respiration the face grows bluish, the lips become livid, the extremities cold, and the body-temperature reduced. There are two poisons in the blood—first, the morphin, and then with it, the carbon dioxid of suffocation. The surface is usually moist. There is often retention of urine. The stupor becomes deeper. The pulse,

which has been full but growing slower, finally becomes weak. Generally at the last the pupils dilate.

There are wide diversities of symptoms. Sometimes convulsions occur in the later stages, more often with children, and in some cases the convulsions have been of a tetanoid character.[1] Sometimes diarrhea occurs with the vomiting. Vomiting is said to be more liable to occur with opium than with morphin, but it is not uncommon with the latter, and as nature's own treatment, it has saved many lives. As a rule, the secretions other than that of the skin are checked. The period when the cerebral symptoms commence, as well as their character, varies greatly in different individuals. There is generally confusion of intellect rather than delusions, and marked delirium is not common. There has been much disagreement as to the uniformity of the contraction of the pupils. Undoubtedly this disagreement has arisen mainly from neglect as to what period of the attack it was when the pupils were observed. It is only in the last stage that the pupils are generally dilated. Certainly there are cases in which there is no contraction of the pupils, but they may fairly be considered quite exceptional. In the early stages, while the pulse is full, the surface is generally warm and often dry, and the dryness of the skin continues throughout in some cases, but in other cases profuse perspiration attends the later stages, and generally the surface is clammy when the pulse begins to fail in strength. When a poisonous quantity is given in successive portions, the second following during the effects of the first and so on, the symptoms of the first stage are extended, and the results, especially upon the respiration, depend upon the rate of elimination of the poison, in proportion to the individual susceptibility and the quantities taken.

A peculiar relapse has marked some cases, in which, after the symptoms have abated and the person has conversed readily for a time, he then falls rapidly into coma and dies.

In differential diagnosis of morphin poisoning it is observed that, as a rule, it differs from alcohol narcosis in the contraction of the pupil; from cerebral hemorrhage, in the two pupils being alike in contraction; from carbolic poisoning, in showing no white stain in the mouth; and from the narcosis of chloroform or ether, in absence of the odor of these agents on the breath. In whatever diseases and from whatever poisons there are failure of respiration and excessive venosity of the blood, there are certain grave symptoms which are likewise found in morphin poisoning. Then the history of the case must be in part relied upon for a differential diagnosis, and, if possible, the question must be settled by a chemical analysis of the liquid vomited or drawn from the stomach.

Chronic Poisoning by Morphin or Opium.—It is scarcely within the scope of this work to discuss the "opium habit." Besides ordinary cases of adults who take the drug by the mouth or hypoder-

[1] On many of the lower animals morphin affects mainly the spinal cord (further see Kobert. *Intoxikationen*, p. 552). Dr. Taylor states that he has found no record of any cases of persons having full tetanic symptoms from morphin poisoning (*On Poisons*, third Amer. ed., p. 647).

mically there are the more obscure cases of infants habitually dosed with nostrums containing morphin,[1] and of persons suffering chronic poisoning due to the continued application of an opiate dressing to a diseased surface. The subjects of the opium habit, though very well known to be able to bear without injury large quantities of the poison—ten or more times as much as an ordinary fatal dose—yet sometimes fall as the victims of acute poisoning by the same drug.[2] The opium eater may exceed his own limit so far as to forfeit his acquired toleration, or this toleration may give way under unusual conditions of the system.

Period when Fatal.—The duration of fatal acute poisoning by morphin or opium, in a considerable proportion of the cases, is from six to twelve hours. It is stated by Woodman and Tidy that death has taken place as early as forty-five minutes and has been delayed as late as four days after the poison was taken. "Of forty-one fatal cases, thirty-one died in times varying from five to eighteen hours, about one-half in from six to ten hours, the most usual time being about nine or ten hours after the poison had been taken. If a patient survives forty-eight hours, the prognosis is favorable."[3] The period when fatal depends little, if at all, upon the quantity of poison taken.

Fatal Quantity.—It may safely be said that 2 grains (0.13 gm.) of morphin sulphate is so poisonous a quantity when swallowed as undoubtedly to endanger the life of a person not accustomed to the drug. Professor Walter S. Haines says: "The average minimum fatal dose of opium for the adult may be placed at about four or five grains, and that of morphin at about one grain."[4] However large a proportion of persons would recover without treatment from the effects of a quantity no larger than this, it could not be expected that every person would so recover.

Children are relatively more susceptible to morphin poisoning than to the action of most other poisons. The difference in the resisting power of adult persons lies especially in the unequal effect of the poison upon the respiratory nerve-centers, and therefore upon the function of respiration, which is the chief natural means of recovery. (For record of some of the smallest quantities known as yet to prove fatal, see Cases 1 to 12.) E. R. von Hofmann states the fatal quantity for average adults to be from 3 to 6 grains (0.2 to 0.4 gm.) of morphin.[5]

A quantity of morphin just above the limit of a medicinal dose in

[1] The sale of medicines offered to the public at large containing such poisons as morphin, without any statement or acknowledgment of the presence of the poison by name, surely ought to be punished by law, either through the application of existing statutes or by the enactment of more specific statutes. Indeed, it seems to me that all *medicines* offered and sold to the public at large should be required by law to have labels giving plainly the name of such medicinal or poisonous agents as they actually contain (A. B. Prescott).

[2] Compare p. 305 in section on General Principles of Toxicology.

[3] *Forensic Medicine and Toxicology*, p. 335.

[4] Hamilton's *System of Legal Medicine*, vol. i., p. 441.

[5] *Gerichtl. Med.*, 1895, p. 690. Kobert (*Intoxikationen*, p. 554) remarks that, in the hypodermic way, morphin has from one to three times the strength and rapidity that it exerts when taken by the mouth.

any case is really on the border of *a poisonous quantity* in that case.[1] Given a barely poisonous quantity,—that is, the smallest one "tending to produce death,"—it will depend on a great number of factors just how much more would be enough to carry out the *tendency* to its fatal conclusion. As to what is the maximum medicinal dose, the physician will be governed in part by the nature of the disease with which he is contending in a given case. For persons of the average susceptibility, or that of people at large while in health, the limit of a medicinal dose given by the mouth has been set by the pharmacopeias of several countries as follows :

MAXIMUM MEDICINAL QUANTITIES OF MORPHIN OR ITS SALTS FOR ADULTS.

	In one dose.		*During twenty-four hours.*	
	Grains.	Grams.	Grains.	Grams.
German Pharmacopeia (1895) . . .	0.5	0.03	1.5	0.10
" " (1872) .	0.5	0.03	1.9	0.12
British Pharmacopeia (1885). . . .	0.5	0.03		
Austrian Pharmacopeia	0.5	0.03	1.9	0.12
Swiss Pharmacopeia (1893)	0.5	0.03	1.5	0.10

MAXIMUM MEDICINAL QUANTITIES OF OPIUM FOR ADULTS.

	In one dose.		*During twenty-four hours.*	
	Grains.	Grams.	Grains.	Grams.
German Pharmacopeia (1895) . . .	2.3	0.15	7.7	0.50
British Pharmacopeia (1885) . . .	3.0	0.20		
Austrian Pharmacopeia	2.3	0.15	7.7	0.50
Swiss Pharmacopeia (1893)	2.3	0.15	7.5	0.50

In Germany the physician must not prescribe quantities above the limit of the pharmacopeia without affixing the sign (!).

Cases are well known of an unusual susceptibility to the action of morphin, so that the person having this idiosyncrasy, as it is termed, suffers poisoning effects from doses not above medicinal limits (see Cases 8, 9, 17).

Treatment of Poisoning by Morphin or Opium.—The chief remedial measures are : (1) To remove the poison from the stomach ; (2) to arouse the patient to breathe ; (3) the permanganate treatment, if we accept recent testimony ; (4) atropin administration as physiologic treatment, urged by many physicians and opposed by some ; (5) strychnin, caffein, strong coffee or tea, cocain, and measures to improve the circulation.

1. To *remove the poison from the stomach* is the first requisite. Generally a simple siphon-tube for the stomach is the best instrument, but a stomach-pump may be used, washing out the stomach two or three times with an abundance of warm water. This should be done even if the poison has been taken in the hypodermic way, because of the abundant discharge of morphin into the stomach from the blood (see Case 23). For the same reason it is well to repeat the washing out of the stomach from time to time. It is most desirable, also, to test the discharges and washings of the stomach for morphin from time to time in order to

[1] "We may, however, conclude that 4 grains (0.26 gm.) of opium and 1 grain (0.06 gm.) of a morphin salt would, in most cases, prove poisonous doses to an adult" (Woodman and Tidy).

learn when the poison ceases to appear in the stomach. If sufficient vomiting result from the poison, each act should be followed by copious drafts of warm water. Emetics are employed to the same end, and must be depended upon if opium has been taken in a mass such as might fail to pass the tube. Ground mustard, in teaspoonful doses repeated, or sulphate of zinc, in doses of from 20 to 30 grains (1.2 to 1.9 gm.), is the most suitable emetic. For hypodermic use apomorphin in doses of $\frac{1}{10}$ of a grain (0.006 gm.) has been employed. To lessen the solubility of the morphin or opium remaining in the stomach and intestines (including the morphin discharged into the stomach from the blood), tea, tannic acid, finely powdered charcoal suspended in water, or iodin in aqueous solution of potassium iodid may be administered. No effective "precipitation" of the alkaloid can be accomplished in the abundant and complex liquids of the stomach. After the danger is averted a saline laxative or enema should be given.

2. To *arouse the patient to breathe*, stimulation of the respiration and of muscular movement is a necessity.[1] Constant urging to effort in breathing at a good rate and application of the magneto-electric current to the spine and chest, or the galvanic brush to the surface, are measures that can be carried on together. Other means are flagellations by cold wet cloths to the head, face, and back of the neck, pinching of the skin, and ammonia to the nostrils. The patient may be kept walking between two assistants. Artificial respiration, about eighteen times a minute, is a resource not to be neglected when breathing fails. Whatever the respiration responds to must be applied with perseverance to the very last, and whatever remedies are employed, the rate of respiration obtained will serve as a measure of their efficacy.[2]

3. The *treatment by potassium permanganate*[3] is advocated as a means of the oxidation of morphin[4] (see Cases 25 to 34. As to the chemistry of morphin oxidation, see p. 497). When given by the mouth, the permanganate is used in doses of about 1 grain (0.06 gm.) more than the quantity of morphin taken, the remedy being dissolved in 30 or 40 parts of water (or 10 grains—0.65 gm.—in a half tumblerful). Moor recommends administration by the mouth, and also hypodermic administration in a saturated aqueous solution, but Wood and Luff[5] do not advise the hypodermic use. Sharp[6] concludes that the action of this agent in the blood of animals is physiologic instead of chemical.

[1] In poisoning by this agent if sufficient respiration can be kept up until the poison is disposed of, all will be well. It is by respiration that, without treatment, more or less of the poison is probably converted into inert oxidation products of morphin.

[2] George L. Peabody, in Foster's *Prac. Ther.*, 1897, vol. ii., p. 43: "The state of the pulse is valueless as a guide." The sole use of the faradic current is to stimulate respiration.

[3] William Moor, *Med. Record*, New York, 1894, vol. xlv., p. 200, and articles following in same journal; *Brit. Med. Jour.*, 1895, p. 1369; H. C. Wood, *Univ. Med. Mag.*, Philadelphia, 1894, vol. vi., pp. 747–752; A. P. Luff, *Brit. Med. Jour.*, 1896, p. 1193; L. Sharp, *Therap. Gaz.*, 1895, pp. 561, 732.

[4] The inhalation of oxygen has been proposed and used as a remedy in poisoning by morphin, but has not justified expectations. Crequy, *Bull. Gén. de Ther.*, Feb. 15, 1880; Limousin, *Amer. Jour. Med. Sci.*, 1880, p. 297.

[5] *Loc. cit.*

[6] *Loc. cit.*

Moor advises that a very dilute solution—1 grain (0.06 gm.) to a tumblerful of water—be administered by the mouth during recovery, to act upon the morphin which is being discharged into the stomach.

4. *Atropin Administration.*[1]—As a respiratory stimulant, and not wholly a physiologic antidote to morphin. Atropin sulphate should be given, the earlier the better, in hypodermic doses, beginning with from $\frac{1}{60}$ to $\frac{1}{20}$ of a grain (0.001 to 0.003 gm.) at discretion, and according to the intensity of the effects of morphin, repeating as necessary to dilate the pupil and then to improve the respiration (see Cases 40 to 57). Tincture of belladonna by the mouth is not so well governed, but may be given beginning with 30 minims (2 c.c.).

5. *Strychnin; Caffein; Strong Coffee or Tea; Cocain; Measures to Improve the Circulation.*—Strychnin in such doses as $\frac{1}{32}$ of a grain (0.002 gm.) hypodermically is advised by H. C. Wood as a respiratory stimulant, more especially when atropin is used. Strong coffee is a common remedy, and caffein hypodermically with equal quantity of sodium salicylate to dissolve in twice its weight of water is a more effective form. Cocain hydrochlorate is indicated in many cases, in hypodermic doses, of from $\frac{1}{4}$ to $\frac{1}{2}$ of a grain (0.016 to 0.032 gm.). The extremities should be warmed, as indicated, using foot-baths, mustard applications, friction, etc.

Cases of Poisoning by Morphin or Opium.

Case 1.—A pill containing 1 grain (0.06 gm.) of the acetate of morphin, taken by a woman, caused narcotic symptoms in half an hour and death in nine hours.[2]

Cases 2-6.—Five cases in which 1 grain (0.06 gm.) of hydrochlorate of morphin proved fatal to adults.[3]

Case 7.—Three grains (0.19 gm.) of morphin taken by a mulatto of sixteen years caused death in twelve hours.[4]

Cases 8 and 9.—According to Christison, 7 drops of laudanum always narcotized a certain gentleman. Half a grain of opium (0.03 gm.) caused narcotism in a woman seen by Grisolle.[5]

Case 10.—A poultice containing much laudanum applied to the pit of the stomach of a young man caused narcotism and death, although treatment was employed.[6]

Case 11.—Five drops of laudanum injected into the rectum of a child of eighteen months caused death in six hours.[7]

[1] Kobert (*Intoxikationen*, 1893, p. 557) has himself used atropin in treatment of opium poisoning for fourteen years, following von Grafe and others. He uses $\frac{1}{60}$-grain (0.001 gm.) hypodermic injections every thirty minutes until the pulse improves and the pupils enlarge. H. C. Wood (*Boston Med. and Surg. Jour.*, 1893, vol. cxxviii., p. 637; Lecture before Harvard Medical School) uses atropin as a respiratory stimulant, combining its effect with that of strychnin. C. H. Lewis (*Detroit Lancet*, 1879, vol. iii., p. 193; reports of cases) uses atropin beyond the dilatation of the pupil to improve the respiration and the pulse, regardless of a resulting soporific state.

[2] *Phar. Jour. Trans.*, July, 1872, p. 16.

[3] With details by Taylor in his work *On Poisons*, p. 549; see *Lancet*, 1872, vol. ii., p. 24.

[4] *Amer. Jour. Med. Sci.*, 1867, p. 562.

[5] Wharton and Stillé, *Med. Juris.*, 1884, p. 341.

[6] Stillé, *Mat. Med.*, vol. i., p. 671.

[7] J. B. Jackson, *Amer. Jour. Med. Sci.*, 1854, p. 384.

CASE 12.—Opium, $\frac{1}{20}$ of a grain (0.003 gm.), caused the death of a child sixteen days old.[1]

CASE 13.—Morphin, 12 grains (0.78 gm.), was taken hypodermically by a girl of nineteen. Respiration was very gravely affected, pulse not affected so much; there was blueness of the face, the pupils contracted to a point, and there was no sensitiveness of the conjunctiva for some hours. Recovery.[2]

CASE 14.—Eight ounces (249 gm.) of crude opium were taken by a pregnant woman of thirty-two years. An hour afterward she was able to give a connected account, and copious vomiting was induced by an emetic. Stupor followed, and later violent burning pain in the stomach. Recovery.[3]

CASE 15.—Crude opium, 180 grains (11.66 gm.), taken by a woman. Symptoms: semicomatose state; slow and feeble pulse; cold skin; relaxed limbs; contracted pupils. Treatment: emetics, strong hot coffee. Recovery.[4]

CASE 16.—Seventy-five grains (4.86 gm.) sulphate of morphin taken by a youth of nineteen years. Symptoms began an hour and a half after swallowing the poison, with sleepiness and a staggering gait. Free emesis then obtained by emetics. Patient then became unconscious, respiration slow and labored, pulse soft and frequent, pupils contracted "to the size of a pin's point." Remedies, belladonna extract, the cold douche, galvanism. Recovery.[5]

CASE 17.—*External application* of about a grain (0.065 gm.) of morphin to a blistered surface at the back of the neck. Patient an aged lady. Symptoms appeared after two hours, consisting of convulsive agitations, cold sweats, extreme prostration, and threatened suffocation. Slow recovery under active treatment.[6]

CASE 18.—Three grains (0.19 gm.) of morphin *injected into the rectum* of a man of forty years. Symptoms, deep coma and contracted pupils. After ten hours the jaws were found so contracted that they could hardly be opened. There were no convulsions. Treatment, instituted after ten hours' delay, consisted of caffein hypodermically and strong coffee by the mouth. Death occurred in sixteen and a quarter hours.[7]

CASE 19.—Half an ounce (14.8 c.c.) of laudanum taken by a man of sixty-two years. Symptoms delayed twelve hours, then complete coma, with contraction of the pupils. Treatment with stomach-pump, coffee, and galvanism. Recovery.[8]

CASE 20.—Laudanum, 1½ ounces (44 c.c.), taken by a man of seventy-two years. Symptoms delayed nine hours, then vomiting, after which the pupils were dilated. Recovery in forty-eight hours.[9]

CASE 21.—Two and one-half ounces (74 c.c.) of liquor opii sedativus (Battley's) taken. Total insensibility followed in fifteen minutes; death, in one hour and twenty minutes.[10]

CASE 22.—Symptoms of strychnin poisoning, convulsions, partial opisthotonos, no coma, followed ¼-grain (0.016 gm.) doses of acetate of morphin. Great relief afforded by hydrocyanic acid. Recovery. Morphin found in the urine.[11]

CASE 23.—Ten grains (0.65 gm.) of opium taken by a Chinaman with fatal result. Seven and a half hours after the poison was taken the stomach was washed out, when the washings responded to tests for morphin and for meconic acid. The urine, being drawn by a catheter, gave tests for morphin. From ten to thirteen and a half hours after the poison was taken the washings of the stomach at intervals gave tests for morphin, but gave negative results in test for meconic acid. It is the conclusion of the observer that this morphin must have been discharged from the blood into the stomach.[12]

[1] E. Smith, *Amer. Jour. Med. Sci.*, 1854, vol. xxviii., p. 381, report of Wm. Morland on "Powerful Effects from Small Quantities of Opium."

[2] *Lancet*, London, 1894, vol. i., p. 669.

[3] *Amer. Med. Recorder*, vol. xiii., p. 418.

[4] J. G. S. Carghil, *Brit. Med. Jour.*, 1879, vol. i., p. 982.

[5] W. F. Norris, *Amer. Jour. Med. Sci.*, 1862, p. 395.

[6] *Amer. Med. Intelligencer*, vol. ii., p. 13; Wormley's *Microchemistry of Poisons*, second ed., p. 479.

[7] Anstie, *Med. Times and Gaz.*, 1863, p. 134. [8] Sloan, *ibid.*, 1855, p. 445.

[9] Gibb, *Amer. Jour. Med. Sci.*, 1858, p. 288.

[10] Beck's *Med. Juris.*, vol. ii., p. 792.

[11] Shearman, *Med. Times and Gaz.*, 1857, p. 285.

[12] L. P. Hamburger, *Johns Hopkins Hosp. Bull.*, 1894, vol. xlii., p. 94.

CASE 24.—A woman of forty-nine years had taken about an ounce (28 c.c.) of laudanum. Was perfectly comatose, with stertorous breathing, feeble pulse, ghastly countenance, pupils "at a pin's point," and full muscular relaxation. Under treatment with emetics, flagellation, and coffee recovery occurred.[1]

CASES 25–28.—A man aged fifty-seven took an unknown quantity of morphin. Found in collapse, body cold, face almost purple, pupils contracted, reflexes absent. Death occurred in twelve hours.

A man of twenty-two years took three teaspoonfuls of morphin sulphate. The respirations were shallow and not over 2 a minute, the pulse slow and full, the pupils narrowed to a pin's point, the face cyanosed, the forehead cold and wet with perspiration. Treatment with permanganate. Recovery.

A woman of twenty-five years had taken ½ ounce (14.8 c.c.) of laudanum. The respirations were shallow and slow, face cyanosed, pupils contracted. Treatment with permanganate. Recovery.

A mulatto woman of twenty-one years took 2 fluidrams (7.4 c.c.) of laudanum with the same volume of glycerin. Respiration and heart action but slightly affected, face cyanosed, pupils "at a pin's point." Treatment with permanganate. Recovery.[2]

CASE 29.—A man had taken 2 ounces (59 c.c.) of laudanum. Was unconscious, the pupils contracted to the size of a pin-point, the respiration scarcely more than a gasp, the pulse very weak. Treatment, potassium permanganate in hypodermic administration. Recovery.[3]

CASE 30.—A woman had taken morphin. Was in profound stupor, with respirations 3 a minute, pulse rapid and irregular, countenance livid, extremities cold. Permanganate solution was injected into the arm. Recovery.[4]

CASE 31.—A woman of middle age had taken a teaspoonful of morphin. Was in a comatose state. The permanganate treatment was given hypodermically. Recovery.[5]

CASE 32.—A child of seven years, not in good health, had taken 2 fluidrams (7 c.c.) of laudanum. Was found with respirations 8 a minute, pulse 46 and unsteady, face pallid and shrunken, expression agonized, and skin cold and covered with sweat. Under the permanganate treatment hypodermically the patient recovered.[6]

CASES 33, 34.—The permanganate treatment, followed by recovery.[7]

CASES 35, 36.—Two cases, a man of thirty-seven and one of fifty-six years, the one having taken 12 grains (0.78 gm.), the other 30 grains (1.94 gm.), of morphin, in each case the respiration nearly failing, were treated with forced respiration and hypodermics of permanganate, with recovery as a result.[8]

CASE 37.—A man had taken 16 grains (1.04 gm.) of morphin sulphate. He was comatose, the respirations were shallow but not decreased in number, the pupils were narrowed to a pin-point, the skin was cold and clammy, and the urine was suppressed. The treatment included the permanganate solution by the mouth and strychnin hypodermically. Recovery.[9]

CASE 38.—A youth of eighteen years had taken 20 grains (1.3 gm.) of morphin sulphate. He was comatose, with respirations 4 a minute, and pupils "at a pin's point." The treatment was by atropin and strychnin hypodermically, and permanganate both by the mouth and hypodermically, and was followed by recovery.[10]

CASE 39.—A boy of seventeen years had taken 6 grains (0.39 gm.) of morphin sulphate. Was cyanosed, with respirations only abdominal and 40 a minute, pulse 160, and temperature 101° F. In treatment atropin and coffee were used without effect. Then *nitroglycerin*, $\frac{1}{50}$ of a grain (0.0013 gm.), was given hypodermically, repeated in an hour. Recovery.

[1] J. D. T. Beckett, *Lancet*, London, 1880, vol. ii., p. 654.
[2] W. L. Pyle, *Med. News*, Philadelphia, 1894, vol. lxiv., p. 514
[3] C. E. Johnson, *ibid.*, Philadelphia, 1894, N. S., vol. xxxii., p. 104.
[4] J. S. Carpenter, *ibid.*, Philadelphia, 1894, vol. lxiv., p. 699.
[5] T. J. M. Lindsay, *Med. Record*, New York, 1895, vol. xlviii., p. 858.
[6] C. H. Callender, *ibid.*, New York, 1894, vol. xlvi., p. 345.
[7] Ebert and Putnam, *ibid.*, New York, 1895, vol. xlvii., pp. 301 303.
[8] J. D. Voorheis, *ibid.*, New York, 1895, vol. xlviii., p. 768.
[9] *Brit. Med. Jour.*, London, 1896, vol. i., p. 82.
[10] A. C. McDonald, *Med. Record*, New York, 1895, vol. xlviii., p. 466.

CASE 40.—A man received 3.7 grains (0.24 gm.) of morphin hypodermically. His respirations were 8 a minute, pulse 40, temperature in the anus 97.2° F. He was unconscious, and a reddish fluid exuded from his mouth. He was treated with *atropin* hypodermically and recovered.[1]

CASE 41.—A man of twenty-eight years had taken 6 ounces (170 gm.) of opium. He was comatose, with pupils contracted to "a pin's point," and pulse slow and full. Treatment: atropin, artificial respiration, and the faradic current. Recovery.[2]

CASE 42.—A woman had taken by mistake a half-teaspoonful of morphin sulphate. Fifty-five minutes later moderate emesis had just been obtained, and soon after the stomach was well washed out. Atropin sulphate was given hypodermically—at first, $\frac{1}{24}$ of a grain (0.0027 gm.); then at intervals of fifteen minutes $\frac{1}{16}$ of a grain (0.004 gm.); and later portions of $\frac{1}{8}$ of a grain (0.0081 gm.). Coffee was administered, coffee fluid extract hypodermically, and frictions and the faradic current were applied. After the fourth injection of atropin the pupils began to dilate, and in an hour covered about one-half of the iris. "They remained at this degree of dilatation and utterly unresponsive to light, with conjunctiva insensible to touch." During the dilatation of the pupils the respirations continued to grow slower and more shallow, the pulse more frequent and feeble, the surface more cold and pale, with increasing sopor. The administration of the atropin salt was continued six hours, and in all $1\frac{1}{16}$ grains (0.0688 gm.) were given subcutaneously. Five hours after the first dose of atropin the respirations were 7 a minute, the pulse 140. At the time of the last administration the respiration and the pulse began to improve, and five hours later the respirations were 14 and the pulse 100 a minute. The period of profound coma was fourteen hours, after which there was recovery without untoward symptoms.[3]

CASE 43.—A woman took from 15 to 20 grains (0.97 to 1.29 gm.) of morphin. The respirations were at one period as slow as 2 a minute, the pupils being contracted to a pin-point. Under hypodermic administration of atropin there was recovery.[4]

CASE 44.—A man of thirty-two years had taken 51 grains (3.3 gm.) of morphin. The respirations were very irregular, the face cyanotic, the lips purple, the pupils contracted to a point. After the poison had been in the stomach thirteen hours the stomach-pump was used along with other treatment—atropin, caffein, galvanism, and flagellations. Recovery.[5]

CASE 45.—A woman of forty-three years had taken 3 drams (11 c.c.) of tincture of opium. The respirations were shallow and irregular, the pulse 90, the pupils contracted, and the patient could not be aroused even with the use of the battery, but rallied under hypodermic administration of atropin.[6]

CASE 46.—A woman of twenty-nine years had taken 2 ounces (56 gm.) of prepared opium. She was comatose, with respirations 4 or 5 a minute. Treatment, atropin hypodermically. Recovery.[7]

CASE 47.—An infant of three months was given a teaspoonful of laudanum. It became semicomatose, there was vomiting, and the pupils were contracted to a point. Two and a half minims (0.15 c.c.) of solution of atropin sulphate were administered with strong coffee. There was recovery.[8]

CASES 48-57.—Ten cases of poisoning by morphin, treatment with atropin, and recovery.[9]

CASE 58.—A woman of twenty-eight years took 16 grains (1.03 gm.) of morphin sulphate and was found in complete narcosis. Under treatment with hypodermic

[1] Kobert, *Schmidt's Jahrb. d. Med.*, 1880, vol. clxxxv., p. 15.
[2] H. H. Taylor, *Lancet*, London, 1884, vol. i., p. 937.
[3] C. H. Lewis, *Detroit Lancet*, 1879, vol. iii., p. 193.
[4] J. H. Smith, *Med. News*, Philadelphia, 1882, vol. xl., p. 318.
[5] G. M. Morse, *Boston Med. and Surg. Jour.*, 1887, vol. cxvi., p. 603.
[6] G. H. Cooke, *Lancet*, London, 1890, vol. ii., p. 1096.
[7] B. L. Paton, *ibid.*, London, 1896, vol. i., p. 548.
[8] H. R. Bramwell, *ibid.*, London, 1889, vol. i., p. 1113.
[9] *Schmidt's Jahrb. d. ges. Med.*, vol. ccxx., p. 22; vol. ccix., p. 138; vol. ccxiii., p. 30; *Amer. Jour. Med. Sci.*, vol. lxxx., p. 578; *Boston Med. and Surg. Jour.*, vol. ciii., p. 297; vol. cv., p. 8; *Med. and Surg. Reporter*, Philadelphia, vol. xlv., p. 533; *Med. Record*, New York, vol. xxix., p. 655; vol. xxiii., p. 487; *Med. News*, Philadelphia, vol. xli., p. 592.

injections of ammonium carbonate and belladonna life was maintained and the patient rallied.[1]

CASE 59.—An infant of one month was given three drops of laudanum. Became semicomatose, and at one time respiration ceased. Artificial respiration with nutrient enemata was employed, and the child recovered.[2]

CASE 60.—A child of fifty-four hours was given ¼ of a grain (0.016 gm.) of morphin. It was found cyanotic, with respirations 3 a minute, and barely perceptible pulse. Under treatment with immersions in hot water, hypodermic injections of whisky, and enemata of strong coffee the child recovered.[3]

CASE 61.—A woman took 92 grains (6 gm.) of opium and became comatose. She was treated with injections of ⅓ of a grain (0.02 gm.) of strychnin sulphate and ⅙ of a grain (0.01 gm.) of atropin sulphate, and recovered.[4]

CASE 62.—A man of twenty-one smoked opium—120 pills containing from 3 to 5 grains (0.19 to 0.32 gm)—daily for two or three days and was poisoned. Respirations 12 or 13 a minute, pulse feeble and rapid, pupils contracted, and face haggard. He was treated with kumiss and brandy, and atropin hypodermically. Partial recovery.[5]

CASE 63.—A man took an unknown quantity of laudanum. The respiration failed at times, but was renewed by artificial means. Pupils strongly contracted and not responsive. Twelve hours after the poison was taken the patient died from heart failure.[6]

CASE 64.—In a case of death by morphin poisoning the autopsy revealed interstitial emphysema as a result of long-continued artificial respiration.[7]

CASE 65.—A child of three years suffering from scarlatina was given about 1 grain (0.065 gm.) of opium, became comatose, and died two hours after the poison was given. The mucous membrane of the stomach was subacutely inflamed. From the contents of the stomach a trace of morphin and meconic acid estimated at $\frac{1}{100}$ of a grain (0.00065 gm.) were obtained.[8]

CASE 66.—A Chinaman of twenty-seven years took 62 grains (4 gm.) of extract of opium. Atropin was used in treatment without much effect. The symptoms were those of morphin poisoning. The autopsy showed hyperemia of the brain.[9]

CASE 67.—The body of a suicide was found two days after death, morphin, strychnin, and copper salt being found in his room. About 8 grains (0.5302 gm.) of morphin were found in the stomach, a small quantity in the intestines, none in the liver or the gall-bladder, and an abundance in the urine, the blood, and the secretions of the mouth and nose, and none in the brain.[10]

Postmortem Appearances.—Morphin poisoning is not distinguished from other modes of death by any peculiar appearances at the autopsy. The blood-vessels of the brain are commonly very full, and frequently with effusion between the brain membranes and into its ventricles. Congestion of the lungs is generally found. The pupils sometimes remain contracted; more often they are of ordinary size or dilated, as, indeed, in some cases they are before death. The blood is usually fluid and dark in color. Upon opening the stomach the attention should be directed toward perceiving any odor—*i. e.*, whether or not it be that of opium. It is only in case of poisoning with poppy preparations that fragments in the stomach are liable to reveal a source

[1] W. C. Coffee, *Med. and Surg. Reporter*, Philadelphia, 1882, vol. xlvii., p. 697.
[2] W. P. Morgan, *Brit. Med. Jour.*, 1888, vol. i., p. 850.
[3] W. Judkins, *Med. Record*, New York, 1885, vol. xxviii., p. 151.
[4] H. C. Wood, *Univ. Med. Mag.*, Philadelphia, 1894, vol. vi., p. 747; *Schmidt's Jahrb. d. Med.*, vol. ccxliii., p. 24.
[5] J. Collins, *Med. Record*, New York, 1889, vol. xxxvi., p. 288.
[6] H. W. Hayes, *Brit. Med. and Surg. Jour.*, vol. ii., p. 807.
[7] M. Mendelsohn, *Schmidt's Jahrb. d. Med.*, 1887, vol. ccxv., p. 144.
[8] J. Priestley, *Brit. Med. Jour.*, 1893, vol. ii., p. 1158.
[9] C. Paster, *Schmidt's Jahrb. d. Med.*, 1887.
[10] Dragendorff's *Organische Gifte*, 1872, p. 130.

of morphin poisoning. No local irritant action on the mucous membrane results from morphin.

Chemical Tests for Morphin.—In the first place the chemist is to keep in mind the distinctive molecular character of this alkaloid. Its well-known avidity for oxidation is not indiscriminate toward oxidizing agents. The first product of its oxidation is oxydimorphin $(C_{17}H_{18}NO_3)_2$, or pseudomorphin. By further oxidation it yields a number of distinctive color-products, undetermined members of known series, variable derivatives of the several color roots of morphin. The color-forming roots obtained as the ultimate chemical fragments of the morphin molecule are morpholin,[1] phenanthrene, and possible derivatives of naphtalene. The synthetic product named by Chastaing "morphin-blue"[2] gives example of these color compounds. There are also a phenolic hydroxyl and an alcoholic hydroxyl, causing other reactive capabilities of morphin, its free solution with fixed alkalis, its picric-acid-like products with nitric acid, its ready conversion to codein and to other derivatives by substitution of acid or alcohol radicles.

When morphin or one of its salts is taken or obtained by itself—that is, nearly or quite free from intermixed matters—it can be identified by tests as follows:

1. **Tests by Iodic Acid.**—There are two very different iodic-acid tests for morphin; these are—(1) The test with iodic acid followed by ammonia (Lefort's test), giving the mahogany color of a morphin derivative; (2) the ordinary test with iodic acid and starch, giving simply liberated iodin and a blue color. The test with iodic acid and ammonia is distinctive of morphin; the test without ammonia, although it has been much more in use and in the books, is not distinctive of morphin, but is a test for any reducing agent of sufficient intensity. The two tests are equally delicate: the former (Lefort's) has positive value in proof of the presence of morphin; the latter (without ammonia) has negative value in proof of the absence of morphin. In both tests the iodic acid acts as an oxidizing agent upon the morphin. The ammonia used in Lefort's test at once decolors the iodin and develops the color of the morphin-oxidized product. Either of these tests may be made upon a spot of dry residue from evaporation, or upon a just perceptible fragment of a solid to be tested; in either case on the white surface of a clean dry porcelain evaporating dish.

For the test distinctive of morphin (Lefort's) add a drop of a solu-

[1] Morpholin has the structure, $HN\left\langle\begin{matrix}CH_2-CH_2\\CH_2-CH_2\end{matrix}\right\rangle O$. It is related to oxazin, $HN\left\langle\begin{matrix}CH=CH\\CH=CH\end{matrix}\right\rangle O$, and phenoxazin, $HN\begin{matrix}C_6H_4\\ \\C_6H_4\end{matrix}O$, the latter being the nucleus of well-known blue and violet coal-tar colors of determined constitution (see Guareschi's *Alkaloide*, Kunze-Krause, Berlin, 1896; Knorr, "On Morpholin as a Synthetic Product," *Pharm. Centralbl.*, 1897).

[2] Chastaing and Barillot, *Compt. rend.*, 1888, vol. cv., pp. 941, 1012; *Jour. Chem. Soc.*, vol. liv., p. 165; further, "Chemical Bibliography of Morphin," *Pharm. Arch.*, 1898.

tion of iodic acid and leave for ten minutes. If the color of free iodin appears, dry and carefully wash off the iodin by floating over it a drop or two of chloroform, repeating until the spot is colorless. Let the residual spot dry, and add a drop or two of ammonia-water of about 10 per cent. strength—enough to leave free ammonia after action. A mahogany color indicates morphin,[1] the color being that of morphin oxidation products. The decanted chloroform solution (itself showing an iodin color if the morphin has been in considerable quantity) may be evaporated for the other test, to the residue a drop of very dilute starch paste being added, when free iodin will be shown by a blue color in the starch.

This negative test may better be made first by itself, thus: To the residual spot on white porcelain add a drop of very dilute starch solution, evaporate to dryness, cool, moisten with the solution of iodic acid from the point of a glass rod, and wait for the blue color. Now, whether or not the blue color of iodized starch be obtained, the positive test may be made at this point by adding a drop or two of ammonia-water, a slight excess, when the mahogany color, at once or after a few minutes, indicates morphin. This form of the test, with the disadvantage of the liberated iodin which has to be taken into colorless combination with sufficient ammonia, has, however, the advantage of avoiding the washing of the residue by chloroform with possible waste of material. Lefort recommends the application of the test as follows: Narrow strips of filter-paper are wet with a concentrated solution of the extracted matter to be tested, the strips dried and wet again several times. The dried strip is then moistened with the solution of iodic acid, partly dried, and then moistened with ammonia-water enough to leave a slight excess of the latter. The mahogany color, extending in the paper as far as it was wet at first, is an evidence of morphin and is quite permanent.

The liberation of iodin from iodic acid is effected promptly by many reducing agents, such as extractive matters from animal tissues or from medicinal drugs, many inorganic compounds, etc. As a reaction of morphin it is highly delicate, so that when the proportions of iodic acid to starch are right, a distinct starch color can be obtained from 0.01 mg.—according to Dupre, from 0.0065 mg. The right proportions of iodic acid and starch should be adjusted by a control test upon morphin, otherwise the test may fail of its full delicacy or even fail altogether.

The test by iodic acid with ammonia (Lefort's test) is distinctive of morphin so far as a single color-reaction can be relied upon. In the laboratory of the writer the application of this test to numerous extracts from various orders of putrefied animal matter, by different modes of extraction, has in no case so far given a fallacious indication. The

[1] J. Lefort, *Jour. Pharm. et de Chim.*, Paris, 1861, vol. xl., p. 97; *Zeitschr. anal. Chem.*, vol. i., p. 134; A. Dupré, *Chem. News*, vol. viii., p. 267; D. L. Davoll, *Jour. Amer. Chem. Soc.*, 1894, vol. xvi., p. 806. Lefort's entire article *Upon Forensic Examination for Morphin* is most valuable.

delicacy of this test nearly equals that of Fröhde's test. Davoll found that $\frac{1}{6400}$ of a grain (0.00001 gm.) of pure morphin would respond unmistakably to the Lefort test, but in the presence of such foreign matter as is almost unavoidable in extraction from tissues or foods he would not claim to recognize less than $\frac{1}{1280}$ of a grain (0.00005 gm.).[1] It is, therefore, somewhat less delicate than the iodic-and-starch test, and the latter has a further practical advantage, in its negative bearing, that the blue is in contrast with common colors of extractive matters.

2. **The Test by Ferric Chlorid.**[2]—For this test the material is best taken in the solid state; a fragment of it, or residue by evaporation of two or three drops of solution several times on the same spot, always on white porcelain, is moistened, from the point of a glass rod, with ferric chlorid solution (1 : 15 or 20). There should be no free acid in the reagent, and it should not be basal enough to be at all turbid. With morphin or its salts a blue color is obtained—a somewhat permanent color—with excess of the reagent becoming greenish-blue (see Plate 7, No. 4). This is one of the least delicate of the tests for morphin. As a reaction it involves the formation of ferrous salts and of oxidation products of morphin, but it is likely that the main color product is a phenolic compound[3] of the unreduced ferric base. Among alkaloids of ordinary occurrence this color-test is distinctive.[4] Great numbers of aromatic compounds having phenolic hydroxyl in their structure with ferric salts give a blue or partly blue color; the homologous phenols, gallic acid, and tannins (the iron-tannin inks), salicylic acid, and wintergreen oil giving like color.[5] The blue color produced by morphin with ferric chlorid is changed to orange fading to yellow by nitric acid, and the same orange changing to yellow is developed upon treating the alkaloid initially with nitric acid.

As for putrefaction products, the writer can only say that in great numbers of tests of the extracts of putrefied tissue to which he has applied the ferric chlorid test he has not obtained therewith the morphin reaction, except in analyses when the presence of morphin was otherwise established beyond question. Even when morphin had been added by the chemist, as in the control analyses of Davoll (p. 456) and Smith (p. 457), the quantity of the morphin in the putrefactive matter used was not usually large enough to respond to this test, owing to its lesser delicacy.

The test by ferric chlorid and ferricyanid of potassium (essentially different from the test by ferric chlorid alone) is merely a reaction of reduction by morphin, the chief or only blue product being ferrous ferricyanid or ferric ferrocyanid or both. These products, containing

[1] *Jour. Amer. Chem. Soc.*, 1894, vol. xvi., p. 807.

[2] Pelletier, *Ann. de Chim.*, 1832, Ser. 2, vol. l., p. 240; *ibid.*, 1836, vol. lxiii., p. 135.

[3] See p. 497.

[4] Pseudomorphin, said to give this test, according to Hesse, is the same as oxydimorphin, the most ready oxidation product of morphin (p. 26); further, as to the structure of oxydimorphin, see Danckwortt (*Arch. Pharm.*, 1891, vol. ccxxviii., p. 572), who claims that this compound contains the four hydroxyls of two molecules of morphin intact.

[5] See the writer's *Organic Analysis*, p. 399.

been added, and here, after the recommended extraction, a clear morphin reaction was obtained. In an investigation by Professor Vaughan (see section on Ptomains and Other Bacterial Products in their Relation to Toxicology, p. 692) the extracts obtained by Dragendorff's method from anaërobic putrefaction of animal matters gave blue colors in Fröhde's test, alike in those portions to which morphin had and those to which it had not been added. In the work of Professor T. G. Wormley in 1890,[1] in the analytic recovery of morphin from the urine of opium patients, from the blood, and from fresh organs of animals, with extraction mainly by amyl alcohol, he depended especially upon the test by Fröhde's reagent.

The sensitiveness of the molybdic and sulphuric acid test enables $\frac{1}{6400}$ of a grain (0.00001 gm.) of morphin, even when slightly contaminated with extractive matter, to give a decisive reaction.

4. **Other oxidizing agents in concentrated sulphuric acid**[2] have been used in color-tests for morphin. Titanic acid instead of the molybdic acid gives a pink-violet to a brown-red color. Concentrated sulphuric acid alone and cold, when free from traces of nitric acid, gives no color to morphin free from traces of other opium alkaloids. On warming by the water-bath, purple to brown colors are developed. In Pellagri's test[3] the portion is first dissolved in hydrochloric acid, then concentrated sulphuric acid is added, and the mixture is evaporated on an oil-bath at 100° to 120° C. The presence of morphin is shown by a purple color at the edges, and after evaporation of all the hydrochloric acid, a red color. Dissolved again in hydrochloric acid it becomes violet when neutralized with sodium bicarbonate. If hydriodic acid is added, the color changes to green and becomes soluble in ether, which it tints purple. In another test, if a trace of nitric acid is added to the sulphuric acid, or if added after warming the alkaloid with pure sulphuric acid, a rose-red color is obtained, soon turning brown—the test of Erdmann and of Husemann. Potassium perchlorate, used as just above directed, gives rise to a deep-brown color (Siebald).

Cane-sugar and sulphuric acid are used in a very delicate morphin test as follows: The residue to be tested is evaporated with from two to eight times as much cane-sugar, and to the dry residue, when cold, a drop of concentrated sulphuric acid is added, the reaction giving a purple color changing to blood red and fading with other colors. Uranium acetate gives a reddish-brown color, and on further addition of alkalis, a deep-red precipitate (Lamal).[4]

5. **Formaldehyd Test.**—The reagent for this test is concentrated sulphuric acid intermixed with an equal volume of the 40 per cent. solution of formaldehyd. The dry residue or material to be tested for morphin, on white porcelain, is touched from the point of a glass rod

[1] Wormley, *Univ. Med. Mag.*, 1890; *Chem. News*, vol. lxii., pp. 65, 79, 99.

[2] Prescott, *Amer. Jour. Pharm.*, 1876, vol. xlviii., p. 62; Vulpius, *Pharm. Centralhalle*, vol. xxxii., p. 231.

[3] G. Pellagri, *Gaz. chim. ital.*, 1877, vol. vii., p. 297; *Jour. Chem. Soc.*, vol. xxxii., p. 808.

[4] *Ibid.*, vol. lxviii., pt. ii., p. 875.

with this reagent. Presence of morphin is indicated by a purple to violet color, fading very slowly.[1]

6. The general precipitating reagents—iodin with potassium iodid, potassium mercuric iodid, potassium bismuth iodid, phosphomolybdic acid, etc.—give quite complete precipitation with morphin. The precipitate by phosphomolybdate dissolves in ammonia with a blue color. The precipitate by fixed alkalis is freely redissolved by their excess; that by ammonia is but slightly redissolved.

7. **The Identification of Morphin Crystals.**—For this purpose the free alkaloid is preferable to its salts, and alcohol of about 95 per cent. is the more favorable solvent. Solution in ammonia-water has been employed, but is not to be recommended. In rapid crystallization, such as occurs in cooling a warm alcohol solution, only needles are obtained—not distinctive. The crystallization should be slow enough to give columns whose end-surfaces will clearly show their boundary-lines. To retard or even prevent altogether the evaporation of the

FIG. 54.—Morphin crystals from alcohol.

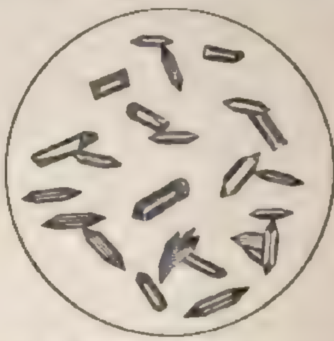


FIG. 55.—Morphin crystals from ammonia solution.

alcohol solution it may be dropped upon a watch-glass, which is then covered with another watch-glass. It serves better still to place the solution on a watch-glass or a glass slide, under a small bell-jar, and place also underneath a small roll of filter-paper wet with alcohol. To obtain crystals from a minute amount of morphin, such as that extracted from tissues or other mixtures, a final residue of such extractions is treated with warm alcohol, in repeated portions of only a drop or two, and the solution, concentrated if need be, is drained into a cavity four or five millimeters wide on a glass surface. A suitable receptacle is made by a section of glass tubing cemented upon a glass slide with a little paraffin. From time to time the result may be observed under a power of 50 diameters, which is suitable for the full examination of crystals. Morphin crystallizes in columns, single and grouped, of the orthorhombic system, with hexagonal end-surfaces. The crystals begin

[1] C. Istrati, *Bul. Soc. Scginte din Bucurcsci*, 1898, vol. vii., p. 168; Prescott, *Phar. Archives*, 1901, vol. iv., p. 89; *Jour. Soc. Chem. Ind.*, 1898, vol. xvii., p. 954; Kobert, *Apoth. Ztg.*, 1899, vol. xxxvii., p. 259

to lose their crystallization water (one molecule) at 75° C.; more rapidly at from 100° to 120° C. The alkaloid blackens before melting and melts at about 230° C. In the subliming cell the upper disc was clouded at 150° C.[1]

The chemist cannot be assured that any crystalline forms are those of morphin unless they are found to respond to the chemical tests for this alkaloid. These tests may be applied to a minute crystal under a good hand-magnifier. All chemical indications gain additional force when obtained from the crystals, however minute.

The Separation of Morphin from Animal Tissues.[2]—The finely divided material, in a weighed portion, is taken in a flask having a return-condenser, covered with diluted alcohol (of about 50 per cent. by volume), distinctly acidulated with acetic acid, and the contents digested in the closed flask at about 70° C. for one hour, when the whole is cooled, strained, the residue washed with a little of the dilute acidulated alcohol, and returned to the flask. On the residue the digestion and straining are repeated a second and a third time. The mixed alcoholic filtrates and washings are evaporated on the water-bath to a syrupy liquid; to this is added three times its volume of very slightly acidulated (acetic) alcohol of 95 per cent., and the whole in a containing flask is corked and shaken and set aside for from twelve to eighteen hours, agitating from time to time.

The mixture is then filtered, using a filter-pump,—better with a Büchner funnel,—and the residue washed with successive small portions of alcohol of the same alcoholic strength and acidulation as the filtrate. All alcohol is now evaporated off at water-bath temperature. Water just perceptibly acidulated with sulphuric acid is now added in quantity barely enough to bring the soluble matter into limpid solution, the whole well stirred, and the insoluble matters filtered out. The filtrate, which must be filtered clear, is now to be shaken out with the immiscible solvents as follows: (1) While acid, with chloroform in several small successive portions; (2) on making the liquid alkaline with ammonia, with four small portions of a mixture of chloroform and ether (3 : 1), acidulating and making alkaline before each portion of the immiscible solvent is used;[3] (3) after slight acidulation and warming, with hot amyl alcohol in several portions; (4) after making alkaline and warming with hot amyl alcohol[4] as the solvent for morphin, this being applied

[1] Blyth, 1878.

[2] This method, as here specified, is but slightly different from that given by Davoll (*Jour. Amer. Chem. Soc.*, 1894, vol. xvi., p. 802) from work done in the chemical laboratory of the University of Michigan. It was also the method used by H. T. Smith, as cited on p. 457. As regards general methods of separation of alkaloids from tissues see p. 330 *et seq.*, in section on General Principles of Toxicology.

[3] The purpose of this solvent is to remove cadaveric alkaloids. In shaking out with each portion the liquid is just acidulated, the immiscible solvent shaken in, then the liquid made perceptibly alkaline, and the mixture at once sufficiently shaken. Generally the shaking-out with an immiscible solvent should be repeated until, after shaking out, a few drops upon evaporation on glass leave no more residue than that resulting from the slight miscibility of the aqueous solution itself.

[4] The amyl alcohol especially should be rectified by redistillation if necessary until the residue of an evaporated portion gives no interfering tests.

in repeated portions. The immiscible solutions, (1), (2), and (3), being respectively chloroform, chloroform-ether, and amyl alcohol, should be once or twice shaken out with a small quantity of acidulated water, and these small water-washings should be added to the aqueous liquid treated in (4). The final morphin extraction in (4) is made with liquid and solvent both hot, and continued in at least three successive portions, and until on evaporating 1 or 2 c.c. a negative result is obtained with one of the more delicate tests. The united amyl alcohol solutions are filtered (first wetting the filter with amyl alcohol), cooled, and shaken out with water in very small portions until the resulting washings give no precipitate with barium chlorid. The amyl alcohol solution, so washed, is measured and held in reserve for qualitative and, if need be, quantitative determinations, each made upon an observed volume.

Portions may be evaporated to dryness for color-tests, and other portions may be shaken out with a little acidulated water for tests by precipitation. It is well to note the volume of each portion taken, in known ratio to the volume of the whole, and it saves time to have the entire or a given part of the amyl solution concentrated to an observed volume. For color-tests a known quantity is evaporated upon the space of a drop or two on white porcelain, dropping the solution in tenths of a cubic centimeter from a pipet only so fast as it dries over the water-bath, and again and again in the same place. Another and a considerable fraction may be evaporated upon glass, taken up in ethyl alcohol, and cooled to crystallize.

If estimation of quantity is to be made, let as large an aliquot part of the amyl solution as is available be evaporated to dryness. In this the morphin may be estimated by the following method: Carefully dissolve all the morphin in the residue by treatment with a very slight excess of dilute acetic acid. Wash and add water to a given volume—say 5 c.c. Take 10 c.c. (or a volume twice that of the morphin solution) of a standard solution of iodin with iodid of potassium, about decinormal in strength (about 1.27 gm. free iodin in 100 c.c.). To this volume of the iodin solution, while shaking, slowly add the measured solution of morphin, add water to make the mixture about five times that of the iodin solution, and shake until the supernatant liquid is perfectly clear or transparent. Take the exact volume of the mixture, filter off just half of this volume (representing exactly half of the iodin solution taken), and titrate with a standard solution of thiosulphate to estimate the free iodin. In this way find the weight of iodin *consumed* in precipitating the alkaloid, and multiply by 0.74918 and by 2, when the product will be the weight of anhydrous morphin in the entire morphin solution precipitated. The precipitate is $C_{17}H_{19}NO_3.HI.I_3$. Then $3I : C_{17}H_{19}NO_3 :: 1.0 : 0.74918$.[1]

This method may also be employed in the estimation of the morphin in mixtures which may have been taken with poisonous effects.

As to the factor of inevitable waste of this alkaloid in extraction from

[1] This method corresponds with that reported by Gordin and Prescott, *Jour. Amer. Chem. Soc.*, 1898, vol. xx., p. 728.

tissue-substances, foods, or medicines, it is best determined by a control analysis, carried with material as nearly as possible the same as that of the real analysis undertaken (see p. 455).[1]

Meconic Acid.—Existing in combination with morphin, in opium, and not known of any other source, the detection of this acid in analysis is important as proof of opium. If both morphin and meconic acid are found, the evidence of opium is complete. Chemical tests for meconic acid are distinctive and delicate. As applied to the contents of the stomach, if opium or laudanum is retained in this organ, the tests for meconic acid offer a reasonable probability of its detection.

Detection.—The contents of the stomach, or other materials analyzed for meconic acid, finely divided, are digested with water moderately acidulated with acetic acid, on the water-bath, for about an hour. The whole is then filtered, the filtrate evaporated to a concentrated solution, three or four volumes of strong alcohol slowly stirred in, and the precipitated matters filtered out and washed with alcohol of about the same strength as the filtrate. The alcohol should now be evaporated from the total filtrate, and the syrupy remainder dissolved in water slightly acidulated with acetic acid and filtered. To the filtered liquid lead acetate solution is added in slight excess. The precipitate, if obtained, is to be examined for the meconic acid as a lead salt. The filtrate and washings are treated with hydrogen sulphid to precipitate all the lead, which, after standing an hour or longer, is filtered out. The last filtrate, with washings, is evaporated to remove all the hydrogen sulphid, and treated, in analysis for morphin, as directed under the head of Separation from Tissues, after the stage of digestion with acetic acid. The precipitate to be examined for meconic acid is now suspended in a little water and treated with hydrogen sulphid gas and filtered to remove all the lead. The filtrate from the lead sulphid is well concentrated by evaporation, filtering to keep clear, and the final solution subjected to the tests for meconic acid. Ferric chlorid strikes a characteristic deep-red or purplish-red color. The color is not readily destroyed by boiling nor by hydrochloric acid (difference from acetic acid), nor by addition of mercuric chlorid (difference from thiocyanic acid). Lead acetate gives a yellowish-white precipitate of lead meconate, not easily dissolved by acetic acid, but colored red when touched by ferric chlorid. Meconic acid is dissolved out from acidulous watery solutions by ether. It

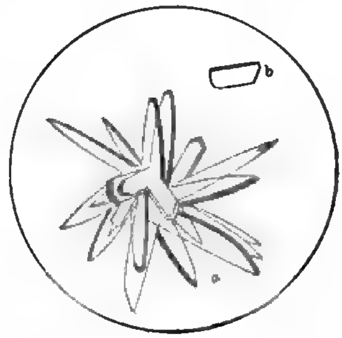


FIG. 56.—Crystals of meconic acid: *a*, From 95 per cent. alcohol; *b*, from water.

[1] See also the article of D. E. Davoll, *Jour. Amer. Chem. Soc.*, 1894, vol. xvi., p. 799. In this article, by inadvertence in the calculation of weight of morphin into that of morphin sulphate (p. 799, A., B., C.), the figure for the sulphate is given at twice the true figure.

is best crystallized in the cooling of a somewhat concentrated hot-water solution or from ordinary alcohol.

In examination for opium the **contents of the stomach** should always be carefully inspected, before chemical treatment, *for fragments of the mass* of opium. Filtered and washed, portions of the sedimentary matters are to be searched by the aid of a hand-magnifier and a microscope with objective of about 50 diameters, comparing with residues of opium similarly treated. On first opening the stomach attention should be given to the odor, whether that of opium or laudanum can be distinguished.

The Deposition of Morphin in the Body.—Morphin is eliminated largely through the mucous membrane of the stomach and intestines, either unchanged or but slightly changed, and only small and varying quantities appear in the urine (Kobert).[1] Its appearance in the alimentary canal as a result of its hypodermic injection is quite marked. (See Case 23 and Case 67; also Analysis 3 and Case 18, under Strychnin.) Wormley,[2] in examining the urine of six persons who had taken morphin, found it present in all the cases, but in four no more than a distinct trace was obtained, such as earlier methods of analysis might fail to reveal. The urine of the remaining two cases yielded considerable quantities of the morphin. That morphin is gotten rid of by oxidation in the blood can hardly be doubted, and the alkalinity of the blood is conducive to the change. The products of the oxidation have not been determined. Morphin itself has been many times obtained from the blood of animals poisoned with it. (See Analysis 5 and Case 67.)

Morphin has been found in the liver, and especially in the gall-bladder and in the kidneys. It has also been found in the brain. (See Analyses 1 and 6).

If the diagnosis of morphin poisoning be in doubt, the urine ought to be examined, if it can be done in time; but failure to find this poison in the urine should not be held as conclusive against its presence in the system.

Analysis 1.—After injection of morphin into the circulation of a cat it was found that the urine, the large intestine, the stomach, and the saliva altogether yielded 12 per cent., and the liver 30 per cent., of the alkaloid administered.[3]

Analysis 2.—Notta and Lugan found morphin constantly in the urine of persons taking this alkaloid habitually, and conclude that it is in these cases largely eliminated in the urine, and can be found when 1½ grains (0.1 gm.) or more are taken daily.[4]

Analysis 3.—On the other hand, Borntrüger could not find any morphin in the urine of a person who took large doses daily by the mouth, but found the alkaloid in the feces. On subcutaneous adminis-

[1] *Lehrbuch der Intoxikationen*, 1893, p. 554.

[2] *Univ. Med. Mag.*, 1890; *Chem. News*, vol. lxii., pp. 65, 79, 99.

[3] E. Marquis, 1896; *Chem. Centralbl.*, 1897, vol. i, p. 249, from *Wien. med. Presse*, 1896, No. 42.

[4] *Chem. Centralbl.*, 1885, p. 967, from *Jour. de Pharm. d'Anvers*, p. 6.

tration, in one person the urine showed traces of morphin even when it was given in very small doses; in another person, to whom much larger doses were administered, the urine showed no trace of the alkaloid.[1]

Analysis 4.—Landsberg concludes, from many experiments with dogs, that, at most, only traces of the morphin reach the urine of the dogs poisoned by it; that a part goes out in the feces and a part is decomposed in the alkaline blood.[2]

Analysis 5.—Tauber found, after injection of morphin into the blood of a dog, that he could recover it largely from the blood and a portion from the feces.[3]

Analysis 6.—A man of forty-two years, a victim of the morphin habit, had formerly taken 62 grains (4 gm.) daily and more recently 30 grains (2 gm.) daily, but had received no morphin for fourteen days, when he died. Morphin was found most largely in the liver, also in the kidneys and in the brain.[4]

Analysis 7.—Wormley, in a few cases, recovered morphin from the blood of animals shortly after administration: "In no instance, however, were crystals obtained or were the results, with perhaps a single exception, such as would have been satisfactory in an unknown case."[5]

In 1890, however, Wormley obtained, by improved methods, more uniform recovery from the blood.[6]

Detection of Morphin in the Body after Long Periods.—Woodman and Tidy recount a case of the recovery and detection of both morphin and meconic acid in the stomach four months after death, the body having lain exposed in an unfinished house.[7] The experiment given on p. 457 shows the detection of morphin ten weeks after the death of the animal poisoned with it. In the experiment with liver, described on page 456, the alkaloid resisted the effects of putrefactive tissue for thirty-five days. Certainly morphin is not to be counted in with the more stable alkaloids; thus its resistance to decomposing effects is far below that of strychnin.

Failure to Detect.—There are many reported cases of failure to find morphin by analysis of the body, even when made shortly after death by poisoning with this alkaloid.[8] In an unknown proportion of these cases, without doubt, the poison would have been revealed by closer chemical work with more efficient chemical method. But there is every reason to believe that *it is not uncommon* in cases of fatal poisoning by morphin for the alkaloid to disappear beyond the reach of analysis—that is, to become so far attenuated that it does not meet the limit of its analytic recovery from the organs in which it lies. In the first

[1] A. Borntrager, *Arch. Pharm.*, 1880, vol. xvii., p. 119.
[2] *Jour. Chem. Soc.*, 1882, vol. xlii., p. 543, from Pflüger's *Archiv*, vol. xxiii., p. 413.
[3] *Chem. Centralbl.*, 1890, vol. ii., p. 606, from *Arch. exp. Path. u. Pharm.*, vol. xxvii., p. 335.
[4] Antheaume and Mouneyrat, *Compt. rend.*, 1897, vol. cxxiv., p. 1475.
[5] *Microchemistry of Poisons*, second edition, p. 512.
[6] *Univ. Med. Mag.*; *Chem. News*, vol. lxii., pp. 65, 79.
[7] *Forensic Medicine and Toxicology*, p. 340.
[8] Taylor, *On Poisons*, third edition, pp. 556, 559

place, it is subject to the result possible to poisons generally—that they may be eliminated from the body too late for the vital powers to recover from the injury they have inflicted. In the next place, morphin is liable to chemical changes and decompositions. The conditions which bring about the decomposition of morphin in the body in one case and not in another are far too complex and elusive to be predicted.

Haines[1] reports a case of analysis of the stomach made for morphin promptly after the death of a woman who had taken from 10 to 15 grains (0.65 to 0.98 gm.) of the drug with suicidal intent. She lived about eighteen hours after taking the poison. After repeated and careful tests not the faintest reaction for morphin could be obtained. The same authority refers to a considerable number of cases in late years, of known death from opium, in which the poison was not afterward discovered in the stomach.

CODEIN.

An alkaloid present in opium in the proportion of 0.2 to 0.8 per cent., its quantity averaging, therefore, about one twenty-fourth of that of the morphin in opium. In chemical constitution it is a morphin derivative,—methyl-morphin, $C_{18}H_{21}NO_3$,—the methyl group being substituted for the phenolic hydrogen of morphin. Codein can be prepared by chemical treatment of morphin. As a medicine, codein has been used more in France than in America, but it was given a place in our pharmacopeia in 1880, and its use in this country is increasing. As a poison, dangerous symptoms have been ascribed to this alkaloid.[2] As furnished, it is liable to contain morphin, if not other opium alkaloids. In effect it is a calmative.

(For **symptoms and treatment** see Cases of Poisoning by Codein, p. 509.)

Tests.—The chemical tests for identification of codein are fairly distinctive when taken together and with observation of the solubilities. In cold concentrated sulphuric acid, free from any trace of nitric acid, it dissolves without color, and on touching this solution with the point of a glass rod moistened with a solution of ferric chlorid (specific gravity, 1.28), a deep-blue color appears. With ferric chlorid solution alone codein does not give a blue color (difference from morphin). The solution of the alkaloid in concentrated sulphuric acid develops a blue color when heated or on long standing. If the solution of the alkaloid in concentrated sulphuric acid be heated to 150° C. and touched with nitric acid, a blood-red color is seen. With Fröhde's reagent codein gives colors—in succession, yellowish, deep green, and finally blue. Codein does not liberate iodin from iodic acid, by which it is clearly distinguished from morphin. It agrees with morphin in decoloring solutions of permanganate of potassium. Codein is soluble in 80 parts of water at 15° C., or in 15 parts of boiling water, in which it melts, and is freely soluble in ether, each solvent serving to separate from

[1] Hamilton's *Legal Medicine*, vol. i., p. 446.
[2] *Brit. Med. Jour.*, 1874, vol. i., p. 478.

morphin. The alkaloid crystallizes from water or aqueous ether in rhombic octahedra or prisms, transparent, melting at 152° C. According to Fouquet (1897), codein is soluble in 16 parts of anise oil at 16° C., morphin being insoluble in the same solvent, from which the codein crystallizes.

The **separation** of codein from contents of stomach, vomited matter, tissues, etc., may be effected by any one of the processes given on p. 330, *et seq.*, in the section on General Principles of Toxicology. If codein alone is looked for, the Stas-Otto process is advantageously used; but if other alkaloids are also sought, the modified Dragendorff process is better.

CASES OF POISONING BY CODEIN.

CASE 1.—A man took 4 grains (0.259 gm.) of codein and in half an hour became much exhilarated in spirits. In four hours he was greatly prostrated, pale, and in a profuse perspiration, with scarcely perceptible pulse and pupils slightly contracted. Vomiting was induced by warm water and gave some relief, after which there was repeated vomiting from the effects of the codein. The second day the patient still suffered from nausea and weakness, but gradually recovered. Afterward he took half-grain doses of codein twice daily for diuresis, as directed by physician, with benefit.[1]

CASE 2.—A hospital patient in advanced phthisis took a quantity of cough-mixture containing about 8 grains (0.518 gm.) of codein, his stomach being empty at the time of taking. In about ten minutes the pulse was 142 and weak, the respiration 30, with sighing. Vomiting was then induced, and whisky given. A few hours later the pulse was 120 and bounding, the pupils were closely contracted, there was a feeling of confusion in the head, and intense itching was present over the arms and trunk. He was kept awake, coffee and ether were administered, and the symptoms gradually abated, with recovery on the second day.[2]

CASE 3.—A young woman who had been taking ¼ of a grain (0.016 gm.) doses of codein on the prescription of a physician took 8 grains (0.518 gm.) at once. An hour later there were nausea and vomiting. The patient then became restless and irritable, making some convulsive movements, and suffering intense irritation of the skin over the body and especially upon the back and the forearms. The respirations numbered twelve a minute, there were thirst and a feeling of fulness in the head, and the pupils were contracted to a pin-point. Under "the usual treatment for opium poisoning" the patient improved, and on the second day recovered.[3]

NICOTIN AND TOBACCO.

Tobacco owes its narcotic and poisonous power, also its chief means of identification, to the volatile and odorous alkaloid, nicotin ($C_{10}H_{14}N_2$).

Pure nicotin is a colorless, oily liquid, slightly heavier than water, slowly soluble by diffusion at ordinary temperatures, exhaling a characteristic pervasive odor, and boiling at 250° C. On prolonged exposure to air it acquires a yellowish color, with gradual resinification. In combustion, as in tobacco smoking, a larger part of it is decomposed—that is, converted into other and simpler pyridin derivatives.

Symptoms and Effects of Poisoning with Nicotin.—Giddiness, prostration, trembling of the limbs, extreme nausea and vomiting, often purging, a feeble and rapid pulse, difficult breathing, the

[1] A. S. Myrtle, *Brit. Med. Jour.*, London, 1874, vol. i., p. 478.
[2] D. Walsh, *ibid.*, London, 1889, vol. ii., p. 718.
[3] W. P. Sprathng, *Medical Record*, New York, 1893, vol. xliv., p. 81.

pupils of the eye variably affected in different cases, but most often dilated, the face pale, the extremities cold, and the mind obscured. Convulsions are not unusual, nor is coma in the last stages. When death occurs, the heart continues to act after respiration ceases. The symptoms begin as soon as a poisonous dose reaches the circulation. External applications of tobacco have caused a number of cases of fatal poisoning. The course of the poison is a very rapid one—measured by minutes more often than by hours. Enemata of tobacco have caused death in fifteen and in eighteen minutes; the pure alkaloid, by the mouth, in five minutes.

According to Kobert,[1] it may be deduced from records and experimental data that 1 grain (0.06 gm.) of nicotin is about the smallest fatal dose for an adult. Tobacco contains from 1.5 to 4.5 per cent. of nicotin.[2] Kobert says a case is known in which 12 grains (0.8 gm.) of tobacco taken internally caused death. Nicotin is excreted by the lungs and the kidneys.

Treatment consists of the evacuation and washing-out of the stomach and the administration, meantime, of finely pulverized charcoal partially to absorb the alkaloid. The poisonous effects are to be met by stimulants, frictions, and warmth, promotion of the respiratory efforts, and the inhalation of oxygen gas.

Postmortem Appearances.—The blood is dark and fluid. Congestion of the brain and visceral organs is usually found.

Chemical Tests for Nicotin.—The odor of the alkaloid is probably the best means of its identification. It will be observed that the odor is not so well obtained when the alkaloid is combined with an acid in a salt as when it is liberated, in this respect behaving like other volatile bases. The vapor of nicotin gives a white cloud with the vapor of hydrochloric acid. If this test is made in the space between a pair of watch-glasses, the upper glass will be covered with an amorphous solid deposit. Mercuric chlorid, with a solution of free nicotin, gives a white precipitate, at first amorphous, but becoming crystalline, the latter characteristic being quite distinctive. The crystals of the nicotin precipitate dissolve in acetic acid. This test is made distinctive from all fixed alkaloids by applying it to a portion of distillate, which may be done by care in an operation with a very small portion of the material. Iodin in aqueous potassium iodid precipitates nicotin in acidulated solutions—a distinction from ammonia. An ether solution of nicotin, treated with an ether solution of iodin, gradually forms ruby-red crystals. Free nicotin is strongly alkaline to indicators. As a divalent base it forms both monacid and diacid salts. In constitution, according to recent researches, it is a pyridyl and pyrrol derivative.[3] It is strongly levorotatory.

The physiologic test, applied to a small animal, is very prompt and decisive.

[1] *Intoxikationen*, 1893.
[2] Allen's *Commercial Organic Analysis*, 1892, vol. iii., pt. ii., p. 192.
[3] Pinner, 1895.

In tobacco the nicotin is in combination with malic and citric acid, both of common occurrence and not easily identified when present in minute quantities. If tobacco has been used in mass, the contents of the stomach or other receptacle may retain shreds of leaf and stem, capable of identification under a careful inspection with a magnifier. When tobacco is used in infusion, the analyst is limited to the identification of nicotin. To the alkaloid is due the odor of tobacco, which, it should be remembered, may be capable of distinct observation when the stomach or other material for analysis is first opened by the chemist.

Separation from Tissues and Other Matters.—The contents of the stomach or portions of an organ of the body are finely divided and digested with water slightly acidulated with hydrochloric acid on the water-bath, for a sufficient time. The mixture is then strained and filtered, and the filtrate together with the washings treated with alcohol, added while stirring. The precipitate produced is removed by filtration; the filtrate and washings are concentrated, then treated with absolute alcohol while stirring, and filtered cold, washing with a little absolute alcohol. The filtrate is evaporated, at a slight elevation of temperature or under the filter-pump, to remove all the alcohol. The aqueous syrupy liquid is made very slightly alkaline by the addition of dilute caustic soda, and then repeatedly shaken out, in a separator, with ether, when the resulting ethereal liquid is allowed to evaporate without heat. If the residue from evaporation of the ether is not sufficiently pure, the treatment should be repeated with acidulated water, absolute alcohol, alkali, and ether, as at first. (As to nicotin-like ptomains, see p. 701 in section on Ptomains and Other Bacterial Products in Their Relation to Toxicology.)

Nicotin remains undecomposed in putrefying tissues for years, being held by union with acids against vaporization. The lungs, the kidneys, and the urine should be examined for it.

Foreign Narcotics in Cigarettes.—Statements have found currency that numerous leading brands of cigarettes contain opium, introduced in manufacture. In order to obtain direct proof as to the truth of this allegation, the writer, in 1889, acting with Chandler, Long, Haines, and other chemists, made quite an extensive and minute chemical examination of certain leading American brands of cigarettes as to the presence of morphin. Analysis was also made for other alkaloids and for meconic acid. No indication of the presence of morphin was found in any instance, nor was any other constituent of opium found, nor any foreign narcotic whatever, in any instance. Testimony to this effect was given by the writer before a committee of the Legislature of the State of Illinois, at Springfield, on March 28, 1893.

Cases of Poisoning by Nicotin and by Tobacco.

Case 1.—The historic case of the trial of the Count of Bocarmé for the murder of his brother-in-law, Gustave Fougnies, has given a degree of forensic interest to this poison; and that it was Stas under whose hand the analysis was made, determining nicotin in the case, has given chemical interest to the records. It was established that nicotin, "the essential oil of tobacco," prepared by the defendant, was forcibly administered, with the result of death in five minutes. In the examination of the body the tongue was found swollen, its epithelium de-

tached, and the blood not coagulated. Nicotin was found in the mouth, throat, stomach, liver, and spleen.[1]

CASE 2.—In London, in 1858, a gentleman swallowed a quantity of nicotin from a bottle, and almost immediately fell to the floor. There were no convulsions. With a deep sigh, life became extinct. This death resembled one caused by prussic acid or potassium cyanid. The appearances were those of relaxation of the muscles, bloated features, and fulness and lividity about the neck. The odor of nicotin or tobacco was not perceptible about the body. Two or three days after death it was found that putrefaction had occurred especially in the course of the veins. There was swelling of the neck, arising from effusion of dark, liquid blood. The scalp and membranes of the brain were filled with dark-colored blood. The lungs were engorged and of a dark purple color. The cavities of the heart were empty, with the exception of the left auricle, which contained 2 drams (7.5 c.c.) of dark-colored blood. The stomach contained a chocolate-colored fluid; the mucous membrane was of a dark, crimson-red color. There was no odor excepting that of putrefaction. The liver was congested. The blood throughout was liquid and dark. Nicotin was found in small quantity in the stomach, also in the liver and lungs, which, however, had been placed in contact with the stomach.[2]

CASE 3.—A man applied to himself a decoction of tobacco for the cure of an eruptive disease. Death took place in three hours, with the usual symptoms of tobacco poisoning.[3]

CASE 4.—A man suffering from pediculi pubis rubbed his entire body with a decoction which he had made by boiling 3000 grains (200 gm.) of tobacco in 2 liters (4 pints) of water. He was seized with vertigo, nausea, heaviness of the head, disturbance of vision, cold sweats, extreme pallor, trembling and weakness of the limbs, etc. The extremities became very cold and purplish in color, and the moisture on the skin was viscous. The pupils were slightly dilated and retained the power of accommodation; they reacted to light. Nausea and vertigo were constant symptoms. There was difficulty of respiration and of speech. The symptoms gradually subsided after three hours.[4]

STRYCHNIN.

General Description.—This well-known poison is an alkaloid obtained from the *Strychnos nux-vomica* and *Strychnos Ignatii*, and is found in other species of Strychnos, a genus of plants of the order Loganiaceæ, in variable proportions reaching as high as 1.5 per cent. With the strychnin ($C_{21}H_{22}N_2O_2$) is found brucin ($C_{23}H_{26}N_2O_4$), a dimethoxystrychnin, of similar but very much milder poisonous effect. Other alkaloids, less widely known, have been found with strychnin, but in very small proportions.

In this country strychnin is usually furnished as the sulphate $(C_{21}H_{22}N_2O_2)_2H_2SO_4.6H_2O$, crystalline in prisms, efflorescent in dry air to a white powder, and soluble in water or in alcohol. It is remarkably bitter. The free alkaloid is colorless, variably crystalline, or occurs in white powder, very slightly soluble in water and moderately soluble in alcohol.

Strychnin is distinguished as an alkaloid of stable composition, resisting various ordinary decomposing influences further than most organic compounds, while still capable of very decisive chemical reactions,

[1] Orfila, *Toxikologie*; a full account of the case is given in Wharton and Stillé's *Med. Jurisprudence*, vol. ii., p. 603.

[2] Alfred Swaine Taylor, *Guy's Hosp. Reps.*, October, 1858, p. 354.

[3] *Amer. Jour. Med. Sci.*, January, 1865, p. 298.

[4] M. Auché, *Amer. Jour. Pharm.*, 1891, p. 463, from *Jour. de méd. de Bordeaux*.

and exerting a markedly characteristic physiologic effect. It is a tetanic poison and affects the muscles rather than the mind.

Symptoms of Poisoning by Strychnin or Nux Vomica.—The symptoms begin in many cases in ten or fifteen minutes after the poison is taken, but they have been known to begin immediately, and in other cases they may be delayed for an hour and even longer (see p. 303 as to causes of difference in the time of onset of symptoms). Usually the first symptoms are a sense of tightness of the chest, stiffness of the neck, slight shudderings, and a feeling of impending calamity, drawing of the hands and feet, and, ere long, quite a sudden and violent tetanic convulsion, in which there are extension of the legs, arching of the feet, with the head drawn back, sometimes so that the body rests upon the heels and the back of the head, the arms extended or drawn tightly across the chest, the face fixed in a grin, the eyes staring, and the muscles of respiration so stiffened that suffocation seems to be threatened. During the paroxysm the pulse is weak and irregular, the countenance is livid, the pupils are dilated, and frequently foam appears at the mouth. The duration of the convulsion is from half a minute to several minutes; the interval until the next convulsion is from a few minutes to half an hour. In the interval the muscular rigidity nearly or quite disappears, there is general relaxation, a cold perspiration covers the surface of the body, and there are contraction of the pupils and a sense of weariness, the patient sometimes falling asleep. The access of another paroxysm is not infrequently preceded by a feeling of anxiety and sensitiveness, and appears to be brought on by any sudden movement or sound to which the patient is exposed. For the most part the mind is clear and steady, although oppressed during the paroxysm. Vomiting is not usual as a symptom, but it has been observed in some cases, as have also pain in the stomach and other signs of irritants.

Strychnin Poisoning Distinguished by its Symptoms.—So distinctive are the usual symptoms in poisoning by strychnin that when they run an ordinary course and have been carefully observed, they can be relied upon quite fully for diagnosis. It is always a necessary inquiry, however, as to whether or not the symptoms observed may be due to some disease. Tetanus especially, also chorea, epilepsy, and uremic and puerperal convulsions, has been claimed to present symptoms liable to be mistaken for those of strychnin poisoning. In regard to these diseases the statements of Walter S. Haines,[1] drawn from very extensive experience, are so valuable that they are here quoted entire: "All of these [the diseases just named] with the exception possibly of tetanus differ so essentially in their manifestations from strychnin poisoning that they could not possibly be confounded by a competent observer. The different forms of tetanus do in some respects resemble strychnin poisoning, yet there are such well-marked differences between them that, after careful examination, they should never be mistaken for each other. In both, it is true, there are violent tonic spasms which are strikingly similar, but in almost all other respects they are unlike. In tetanus we

[1] Hamilton's *Legal Medicine*, vol. i., p. 449.

almost always have a history of an injury, and the disease usually comes on gradually and progresses slowly to a fatal termination, a number of days generally elapsing between the onset of the disease and death, while in strychnin poisoning there is no antecedent injury and the manifestations appear suddenly, almost without warning, and progress rapidly to a fatal termination; in tetanus the muscles first affected are generally those of the back of the neck, and those of the jaw are early invaded, producing persistent lockjaw, while in strychnin poisoning either the muscles of the extremities are first affected, and those of the neck and jaw last attacked, or the entire muscular system is thrown into a spasm almost, or quite, simultaneously; in tetanus there is usually considerable persistence of the muscular rigidity, even between the severe spasms, and opisthotonos or some other perverted position of the body is generally permanent, while in strychnin poisoning there is almost always complete relaxation between the spasms, and the opisthotonos, which may have been very marked during the attack, entirely passes away; in tetanus the temperature is generally somewhat elevated beyond the normal, while in strychnin poisoning it is not usually affected.

"There are, it is true, occasionally marked exceptions to the above differences, one case being recorded in which tetanus proved fatal in twelve hours after the first twitchings, another within an hour and a half after the first convulsion, and a third in fifteen minutes after the injury producing the disease; on the other hand, a case of strychnin poisoning is recorded in which death occurred after a lapse of eighteen hours, and several cases are known in which repeated small doses of the poison have been administered in such a way as to extend the symptoms over a period of several days. So also in tetanus, cases are occasionally seen in which little or no increase of temperature is found, while in poisoning by strychnin the temperature is sometimes elevated. In spite, however, of these irregularities both in strychnin poisoning and in tetanus, a competent observer will rarely have difficulty in distinguishing the two, the difference in the order in which the muscles are attacked and the marked dissimilarity in the condition of the patient between the spasms generally sufficing to establish the exact character of the derangement."

Period When Fatal.—In the majority of fatal cases death occurs in from one to three hours; cases of death in ten minutes and in fifteen minutes and a case of death in eleven hours are given by Haines. If life is prolonged for five or six hours, recovery is probable. Of 57 cases of strychnin poisoning collected by Falck, 35 per cent. were fatal.[1]

Fatal Quantity.—One half of a grain (0.032 gm.) of strychnin or one of its salts is a poisonous quantity and liable to prove fatal. One-fourth of a grain (0.016 gm.) is reported[2] to have caused the death of a woman of thirty-six years in one and three-quarter hours after it was taken. A man of thirty-nine years took ½ of a grain (0.032 gm.) of strychnin sulphate; according to record, symptoms began in five minutes and

[1] Kobert, *Intoxikationen*, p. 664.
[2] *Medical Times and Gaz.*, London, 1854, p. 376.

death occurred in twenty minutes. (See Cases 1 to 12.)[1] Certainly there would be a larger percentage of recoveries from the effects of $\frac{1}{2}$ of a grain (0.032 gm.) of this poison than from the effects of 2 grains (0.129 gm.), irrespective of treatment. But the result of any poisonous dose is governed by the product of a considerable number of factors, of which the dose is but one.[2]

Kobert states that the lethal dose for adults is from $\frac{1}{2}$ to $1\frac{1}{2}$ grains (0.032 to 0.1 gm.); for children, $\frac{1}{16}$ of a grain (0.004 gm.).[3]

Treatment of Poisoning by Strychnin.—1. The stomach should be cleaned out at once by the siphon tube or stomach-pump or by emetics and water-drinking. The muscular spasms often interfere with the introduction of the tube, and the inhalation of chloroform is to be hastened to abate the spasms as soon as possible, so that the stomach can be washed out. Solution of iodin, finely powdered charcoal, or tannic acid may well be given at first to diminish the solubility of the alkaloid while it is being removed from the stomach. Apomorphin hypodermically is advised as an emetic (Kobert).

2. The inhalation of chloroform during the paroxysms and the administration of chloral hydrate by the mouth or hypodermically during the intervals are valuable remedial measures.

3. "The patient should be kept as quiet as possible; all strangers should be excluded from the room, direct drafts of air should be shut off, and loud noises, such as those produced by the slamming of doors, stamping of feet, etc., should be prevented. For the same reason as little medicine as possible should be given internally, as the mere raising of the head or touching of the lips may cause a convulsion."[4]

4. If respiration is much diminished by the spasm, pure oxygen may be added to the air inhaled by the patient. Let it be borne in mind that the course of the poison is a short one, and if the patient can be carried through this period, he will recover.

Cases of Poisoning by Strychnin.

Case 1.—A dose of $\frac{5}{16}$ of a grain (0.02 gm.) of strychnin phosphate was taken by a man twenty-nine years of age. The respiration was almost suspended, the pulse small, soft, and too rapid to count, and the skin warm. The treatment was by $\frac{1}{2}$ of an ounce (14 gm.) of potassium bromid. The patient recovered.[5]

Case 2.—Three-fourths of a grain (0.049 gm.) of strychnin was taken by a woman of fifty years. There were convulsions; the body became perfectly rigid; the pulse was rapid. Under treatment by potassium bromid and chloral the patient recovered.[6]

[1] *Brit. Med. Jour.*, August, 1847; Warner, in Woodman and Tidy's *Forensic Medicine and Toxicology*, 1877, p. 313.

[2] The *average fatal dose* for a man amounts, for internal administration, to $1\frac{1}{2}$ or $1\frac{3}{4}$ grains (0.10 or 0.12 gm.). There are, however, cases in which death has occurred from $\frac{1}{2}$ of a grain (0.032 gm.), while in other cases recovery has occurred after 9 grains (0.6 gm.) or even 19 grains (1.25 gm.). In these cases it is to be remembered, however, that emetics had been given (Schmiedeberg, *Pharmacology*, 1887, p. 17).

[3] Kobert, *Intoxikationen*, p. 664.

[4] These directions of Haines (Hamilton's *System of Legal Medicine*, vol. i., p. 451) are based upon sound pharmacology.

[5] W. B. Caley, *Lancet*, London, 1891, vol. i., p. 18.

[6] E. Prideaux, *ibid.*, London, 1881, vol. i., p. 52.

Case 3.—A dose of 0.77 grain (0.05 gm.) of strychnin nitrate was taken by a man of forty-one years. Symptoms: trismus, convulsions, stiffness of limbs, head bent back, without complete opisthotonos, the pulse 140 to 160, the respiration loud and labored. Treatment: potassium bromid and hypodermic administration of morphin. Recovery.[1]

Case 4.—A dose of $\frac{3}{4}$ of a grain (0.049 gm.) of strychnin was taken by a woman of thirty-five years. There were spasms, pains in the legs, and a semidelirious state. Treated with apomorphin hypodermically and with chloroform inhalation, the patient recovered.[2]

Case 5.—A dose of only $\frac{18}{65}$ of a grain (0.019 gm.) of strychnin arsenate was taken by a man of twenty-one years, with resulting clonic convulsions and opisthotonos. There was treatment with morphin hypodermically and by inhalation of chloroform. The patient recovered in five days.[3]

Case 6.—A woman of twenty-five years took 2 grains (0.13 gm.) of strychnin. There were spasms, extreme opisthotonos, eyes turned up and divergent, the right pupil contracted and the left one dilated, the lower jaw firmly set, the respiration at one time being suspended for a minute and a half. Under treatment the patient recovered.[4]

Case 7.—A man of thirty years took 6 grains (0.4 gm.) of strychnin. He suffered violent paroxysms. Treated with chloral hydrate, both hypodermically and by the mouth, the patient recovered.[5]

Case 8.—A youth of eighteen years took 5 grains (0.325 gm.) of strychnin. Symptoms: convulsions, the face cyanosed, no radial pulse, the eyes blood-shot, the pupils of the eye dilated. Treatment: emetics and chloral hydrate by the mouth. Recovery.[6]

Case 9.—A woman of fifty-six years took 4 grains (0.26 gm.) of strychnin. Symptoms: convulsions, lividity of the face, pulse 100. Treatment: emetics and hypodermic administration of morphin and atropin. Recovery.[7]

Case 10.—A woman of twenty-eight years, poisoned by an unknown quantity of strychnin, suffered spasms with complete opisthotonos, and under treatment with atropin hypodermically, recovered.[8]

Case 11.—A woman of twenty-three years, poisoned by 6 grains (0.4 gm.) of strychnin, suffered convulsions, became unconscious, was treated with hypodermic administration of chloral hydrate, and recovered.[9]

Case 12.—A healthy man of twenty-one years took from 13 to 18 grains of *liquor strychninæ* (Br.) ($\frac{1}{3}$ of a grain—0.022 gm.—of strychnin) upon an empty stomach, and the symptoms which appeared in from fifteen to twenty minutes were clonic paroxysms, a cyanotic face, and dyspnea. Death followed in a short time. At the autopsy the muscles of the upper extremities, the jaws, and the eyelids were firmly contracted; the right side of the heart and the pulmonary vessels were filled with dark venous blood; the left side of the heart was empty; the brain was pale. The fluid in the stomach reacted to the test for strychnin; the urine was not examined.[10]

Case 13.—A man took from 2 to 3 grains (0.13 to 0.2 gm.) of strychnin and died in one hour and a half. At the autopsy the abdominal and pectoral muscles were in a state of extreme tension, the blood was of a very dark color, the brain and upper part of the spinal cord were congested, as were also the meninges.

Case 14.—A man took an unknown quantity of strychnin and died, after symptoms of the poisoning, in fifty-five minutes. At the examination of the body the blood was found to be dark, the brain, medulla, and upper part of the spinal cord were congested, as were also the lungs. About $\frac{1}{10}$ grain (0.006 gm.) of

[1] Prinzing, *Schmidt's Jahrb. d. Med.*, 1890, vol. ccxxv., p. 131.

[2] Masson, *Brit. Med. Jour.*, 1894, vol. ii., p. 808.

[3] P. Bock, *Jour. de Méd. et de Chir.*, 1893, vol. li., pl. vii., p. 97; *Schmidt's Jahrb. d. Med.*, vol. ccxxxix., p. 133.

[4] S. Ott, *Med. News*, Philadelphia, 1894, vol. lxv., p. 270.

[5] H. C. W. Jones, *Lancet*, London, 1889, vol. ii., p. 166.

[6] W. F. Wright, *Boston Med. and Surg. Jour.*, 1881, vol. cv., p. 8.

[7] J. N. Vojé, *Med. and Surg. Reporter*, Philadelphia, 1879, vol. xli., p. 525.

[8] S. Buckley, *Amer. Jour. Med. Sci.*, 1874, vol. lvii., p. 278.

[9] V. Fancion, *Schmidt's Jahrb. d. Med.*, 1884, vol. cciii., p. 20.

[10] P. T. Adams, *Brit. Med. Jour.*, 1894, vol. i., p. 300.

strychnin was recovered from the stomach, and about an equal quantity was recovered from the washings of the stomach obtained during treatment by the stomach-pump.[1]

CASE 15.—A man of seventy-two years took an unknown dose of strychnin, suffered symptoms of the poisoning, and died. Forty-four hours after death there was persistence of rigor mortis, although a good deal of decomposition had occurred; no clots of blood were in the heart, and the thoracic muscles were pale. An abundance of strychnin was found in the stomach, along with 16 ounces (453 gm.) of partially digested food.[2]

CASE 16.—An infant was poisoned with nux vomica; three days after death cadaveric rigidity remained. Chemical analysis failed to reveal strychnin.[3]

CASE 17.—A person died from strychnin poisoning caused by two hypodermic injections of tincture of nux vomica. The brain was examined and strychnin found to be present.[4]

Another person died from hypodermic injections of nux vomica; analysis of the stomach yielded $\frac{7}{11}$ of a grain (0.042 gm.) of strychnin, and again the presence of the alkaloid in the brain was determined.[5]

CASE 18.—A man of twenty-four years, habituated to opium, died from a dose of strychnin. Ipecacuanha was used in the treatment. In the stomach no alkaloid was found; in the small intestine a little strychnin but no emetin or morphin; in the large intestine traces of strychnin, but neither morphin nor emetin; in 1033 grains (67 gm.) of blood from the heart, perhaps a trace of strychnin, but no emetin or morphin; in 2000 grains (130 gm.) of blood from the abdominal cavity a trace of morphin and undoubted traces of strychnin; in the liver, traces of strychnin but no emetin, the test for morphin lost; in the brain, traces of strychnin; in the second vomit, distinct evidence of strychnin, much emetin, but no morphin; in the third vomit, distinct proof of both strychnin and emetin, morphin uncertain; in the fourth and the fifth vomit, morphin distinct, but no emetin or strychnin.[6]

CASE 19.—A person died June 23, 1892, from poisoning by strychnin, as it appeared later. The body was exhumed after three hundred and eight days—that is, on April 25, 1893. Of the remains of the body, 5401 grains (350 gm.) were subjected to analysis for alkaloids—in fact, for strychnin. The final alkaloidal residue was twice purified by action of concentrated sulphuric acid for two hours on the water-bath. The tests for strychnin were obtained. Its quantity was estimated at $\frac{1}{32}$ of a grain (0.002 gm.).[7]

CASE 20.—A cat died fourteen minutes after receiving $1\frac{1}{2}$ grains (0.09 gm.) of strychnin. The alkaloid was found in the stomach, duodenum, upper small intestine, liver, kidneys, and bladder.[8]

Postmortem Appearances.—In a short time after death the muscles become extremely rigid, the joints fixed, the fingers closed, the feet arched, and sometimes the body bent backward. This extreme rigidity continues for a variable period—sometimes for a week and sometimes until after a good deal of decomposition has occurred. The brain and upper part of the spinal cord and their inclosing membranes are usually, although not always, overcharged with blood; the same congestion is frequently found in the liver and kidneys, and sometimes in the mucous membrane of the stomach. The heart is found to vary in respect to the amount of blood it retains (see Case 12). The *rigor mortis* is the most characteristic of the ascribed appearances; the others are not very distinctive nor very uniformly present.

[1] F. E. Marston, *Lancet*, London, 1886, vol. ii., p. 442.
[2] L. Ogilvie, *Brit. Med. Jour.*, 1884, vol. i., p. 1251.
[3] *Amer. Jour. Med. Sci.*, 1879, vol. lxxvii., p. 574.
[4] Grandval and Lajoux, *Jour. Pharm. Chim.*, 1879 (4), vol. xxx., p. 164.
[5] *Ibid.*, 1892; *Repert. de Pharm.*, 1892, p. 304.
[6] Dragendorff, *Organische Gifte*, second ed., p. 191.
[7] W. A. Noyes, *Jour. Amer. Chem. Soc.*, 1894, vol. xvi., p. 108.
[8] Dragendorff, *Organische Gifte*, p. 191.

Chemical Tests for Strychnin.—As already intimated (p. 512), the stability of this alkaloid, together with its decisive chemical and physiologic responses, enables it to be identified in tests of great delicacy.

1. **The Taste.**—The bitterness of strychnin is more intense than that of any other known substance. It can be perceived in a solution diluted to 600,000 or 700,000 parts. Wormley states that a drop of a 1 : 10,000 solution has a decidedly bitter taste, not usually masked by the presence of a very notable quantity of foreign matter.

2. **The Fading Purple Test.**—This test properly depends upon two facts—the one being that concentrated sulphuric acid at temperature of the water-bath purifies strychnin instead of coloring it; the other that the cold solution of strychnin in concentrated sulphuric acid, treated with certain oxidizing agents, gives an evanescent play of bright colors. The alkaloid is taken in solid state and dry, on a white porcelain surface, and the area of the test need not be over one-fourth of an inch in diameter. A film of residue can be obtained in a separate narrow area by evaporating a drop of quite concentrated (chloroform) solution, and, if need be, several times repeating the evaporation on the same spot. The residue (or other solid portion) of the alkaloid is touched with the sulphuric acid at the point of a very narrow glass rod and left to dissolve. A small fragment of potassium dichromate is then brought by the glass rod into the liquid and slowly drawn through it at short intervals. The strychnin reaction is an appearance of changing colors—first blue to purple, then violet to reddish (see Plate 7, No. 5). The oxidizing agents which have been used are potassium dichromate, cerosoceric oxid, manganese dioxid, lead peroxid, and potassium permanganate. Haines prefers cerosoceric oxid or the dichromate. The writer has used the dichromate.[1] H. Letheby recommends the use of the electric current. The operator should make himself familiar with the behavior of the chosen oxidizing agent, both with sulphuric acid alone and with the sulphuric solution of strychnin. The change of color is slower with cerosoceric oxid than with the dichromate. If the residue (or fragment) to be tested has not been subjected to the action of concentrated sulphuric acid upon the water-bath, as directed under Separation from Tissues, p. 521, then the sulphuric solution prepared as above is first heated on a water-bath, then cooled, and tested as above stated. A mere darkening by the sulphuric acid is likely to be due to impurity of non-alkaloidal matter. If not enough to interfere with the color test, such darkening may be disregarded. If there is too much darkening, the portion is treated for the purification of strychnin by hot concentrated sulphuric acid, as directed under Separation from Tissues, and then subjected to the color-test.

A distinctive reaction can be obtained with about 0.000037 or $\frac{1}{27027}$ of a grain (0.0000025 gm.) of strychnin.[2]

[1] See the author's *Organic Analysis* 1887, p. 452.

[2] This limit was found when 1 c.c. of solution of strychnin was evaporated and the residue treated with one drop of sulphuric acid (see *Organic Analysis*, p. 452;

The fading purple reaction of strychnin is prevented by the presence of much brucin or by much morphin. The absence of these alkaloids should be assured in the purification of the alkaloid for strychnin tests, as also the absence of sugar, or tissue substances in more than traces, etc. Solubility in chloroform or other volatile solvent and resistance to hot concentrated sulphuric acid are chief among the measures for purification.

Various substances have been asserted to give nearly or quite the same fading color-reaction as strychnin. The most of these substances are excluded at once by regarding the first condition of the test, the previous action of hot concentrated sulphuric acid, as already stated. As to ptomains in this reaction, see p. 702 in the section on Ptomains and Other Bacterial Products in Their Relation to Toxicology. Hydrastin, which is faintly colored yellow by sulphuric acid, treated with oxidizing agents, gives a play of colors not the same as that from strychnin.[1] Quebrachin is destroyed by heating with sulphuric acid (Haines). Curarin is colored by sulphuric acid, and is not very soluble in chloroform. Gelsemin gives a reddish-purple to cherry-red color.

The Lloyd Reaction of Morphin with Hydrastin in the Fading Purple Test.—In his popular novel, *Stringtown on the Pike*, published in 1900, Professor John Uri Lloyd introduces into the murder trial of Red Head, what has proved a feature of much chemical interest, a fallacy of the fading purple test, due to the combined reaction of two alkaloids, the one contained in golden-seal root and the other in laudanum. The present writer is indebted to his friend, Professor Lloyd, for a fine sample of the mixed alkaloids—hydrastin nine parts with morphin one part. Tests of this mixture confirm results the writer had previously obtained with hydrastin mixed with morphin and with hydrastin alone, in effect as follows: The mixture of hydrastin and morphin, treated on white porcelain with concentrated sulphuric acid and not heated, at first shows only a pale-yellow color, gradually deepening as it stands. On adding a fragment of potassium dichromate there is a play of purple to maroon-red coloration. The colors do not fade, but they grow gradually darker and duller, so that in from fifteen to thirty minutes the color becomes nearly or quite uniform and of a dark brown, varying from the prevailing color of mahogany to that of black walnut wood when oil finished. Hydrastin alone, treated on white porcelain with concentrated sulphuric acid, gains a pale-yellow color, the same result that is obtained with the mixture of hydrastin and morphin. If, now, the heat of a water-bath be applied, as recommended for the purification of strychnin, the liquid soon darkens and deepens, without any play of color, to a maroon-red and then mahogany-brown coloration. Later very dark particles of solid appear, due to the beginning of carbonization by the hot sulphuric

Chem. News, vol. liii., p. 78). Wormley found a limit at $\frac{1}{10000}$ of a grain (0.000007 gm.), and Hinsdale with cerosoceric oxid at $\frac{1}{100000}$ of a grain (0.0000007 gm.). For greatest delicacy the alkaloidal residue should be brought into an area much smaller than that of the residue of 1 c.c. in a single evaporation

[1] A. B. Lyons, *Amer. Jour. Pharm.*, 1886; see also the next paragraph.

acid. This action of hot sulphuric acid enables the operator to remove hydrastin, as well as ordinary tissue substances, from any strychnin in the residues under treatment. Hydrastin itself, the colorless alkaloid of *Hydrastis canadensis*, with the formula $C_{21}H_{21}NO_6$, crystallizes trimetric, and melts at 135° C. It is nearly insoluble in water, very freely soluble in chloroform, soluble in about 16 parts of benzol, about 84 parts of ether, and 120 parts of alcohol.[1]

3. **The Chromate Test.**—When a drop of an aqueous solution of a salt of strychnin is touched with solution of potassium dichromate, a yellow crystalline precipitate can be obtained, and the crystals are octahedral and dendroidal in form. Only the octahedra are distinctive; dendroidal or brush-like aggregations may be formed from the dichromate itself. In applying this test to a portion of the extract from tissues, a few drops of the chloroform solution are evaporated upon a glass slide, the residue taken up with a drop of dilute acetic acid, and the excess of acid mostly dissipated, then the border of the liquid wetted with the dichromate solution and the mixture stirred, without unduly spreading it, at the point of a very narrow glass rod. The slow formation of a crystalline precipitate is promoted by slight concentration or by slight dilution. Any crystals obtained are examined under a microscope with a suitable power, comparing with a control test.

FIG. 57.—Strychnin with the dichromate.

Fading Purple Test of the Chromate.—If the crystals of the chromate be obtained, they are to be gathered to a point on the glass slide and dried, then touched with concentrated sulphuric acid, when the play of fading colors will be obtained the same as in the direct color-test.

The Physiologic Test.—The frog is the animal to be taken. The aqueous solution of a salt of the alkaloid, not more than slightly acidulous, nearly purified when obtained by extraction from tissues or foods, is given to a small frog, either hypodermically or by blowing into the stomach through a small tube. The time is noted and the animal is put under a glass jar and observed. Tetanic spasms are caused by strychnin. Wormley obtained, with frogs of from 15 to 50 grains (0.98 to 3.3 gm.) in weight, from $\frac{1}{5000}$ of a grain (0.000013 gm.), distinctive symptoms in from ten to thirty minutes; from $\frac{1}{500}$ of a grain (0.00013 gm.), symptoms in three or four minutes and death in from fifteen to thirty minutes. Kobert mentions $\frac{1}{30}$ of a grain (0.002 gm.) as a fatal dose for

[1] On the history of hydrastin see a part of an article by the writer, *Jour. Amer. Chem. Soc.*, 1899, vol. xxi., p. 736.

a frog. The paroxysms at first have remissions, and their return is hastened by agitation or vibration, as by striking on the table.

The **melting-point** of strychnin as a free alkaloid is stated to be 265° C. (Beckurts, 1890); 268° C. (Löbisch and Schoop, 1886); 265°–266° C. (Stoehr, 1885). It crystallizes in the isometric system. Rhombic dodecahedrons are prominent figures. A good way to obtain its crystals in characteristic forms for microscopic inspection is gradually to add water to its alcoholic solution. It crystallizes well from benzene.

Strychnin (free alkaloid) is soluble in about 8300 parts of water or 2500 parts of boiling water, 207 parts of absolute alcohol, 140 parts of benzene, in about 1200 parts of ether, in 8 to 10 parts of chloroform, in a moderate amount of a mixture of equal volumes of chloroform and ether, and, according to Wormley, requires no less than 12,500 parts of petroleum ether to dissolve it.

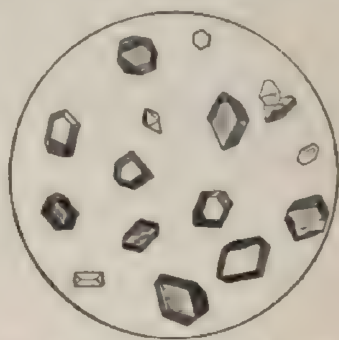


FIG. 58.—Strychnin crystals from benzene.

Strychnin is precipitated from solutions of its salts by the general reagents for alkaloids—iodopotassium iodid, potassium mercuric iodid, phosphomolybdic acid, picric acid, tannic acid, etc., and by the alkali hydroxids, redissolving in excess of ammonia.

Separation of Strychnin from Tissues, Foods, or Contents of the Stomach.—A weighed quantity of the material, or determinate fraction of the organ, finely divided if necessary, with water enough to make it into a free flowing mixture, is strongly acidulated with acetic acid [1] and digested on the water-bath for about an hour, adding water to replace that evaporated. The mixture is strained hot, the liquid concentrated on the water-bath to about one-fourth its first volume, and to this reduced liquid, while hot, five or six times as much strong alcohol is slowly stirred in. A good deal of organic matter is precipitated by the alcohol, and this is to be filtered out, washing the precipitate with portions of alcohol of the same strength as that of the filtrate. The total filtrate is evaporated on the water-bath to a syrupy liquid, and this, when cold, is treated with enough water, slightly acidulated with acetic acid, to make it feasible to filter the whole, quite clear, into a separatory bulb or strong large test-tube, for shaking out with about an equal bulk of chloroform or other solvent immiscible with water.

Directly before shaking out the liquid is made very slightly alkaline by addition of potassium hydroxid solution. The shaking-out is to be done thoroughly, but guarding against making an emulsion, and after drawing off the separated chloroform, the shaking-out is repeated with a second and perhaps a third portion of the chloroform. If the solvent

[1] These directions are mainly those given by Haines, from his large experience, in Hamilton's *Legal Medicine*, vol. i., p. 456.

refuses to separate, ether may be added in volume equal to that of the chloroform, and this ether-chloroform solvent may be taken, in shaking out a second and a third time. Chloroform alone is to be preferred, if it does not emulsionize too much. The united chloroformic portions, obtained quite clear, are gently evaporated in a porcelain dish. The residue, still containing a good deal of extraneous organic matter, will contain the strychnin, if present in the material operated upon. To purify the alkaloid further, the shaking-out with chloroform is repeated by taking up the residue with dilute acetic acid, filtering, adding the chloroform in the separatory tube, making alkaline, and going on as before, again evaporating the chloroform solution in a porcelain dish.

Almost always it is necessary to purify the residue further, and in case of strychnin there is a remarkable opportunity to do this by action of hot concentrated sulphuric acid. To do this, the dry residue is well moistened with sulphuric acid, two or three drops being usually sufficient, and the whole heated on the water-bath for an hour or two.[1] The blackened residue is now stirred with water, filtered and washed, made faintly alkaline, and shaken out with chloroform. The chloroform solution is sufficiently concentrated and then kept under a stopper, preferably in a small graduated cylinder. Small portions are taken with care for evaporation on porcelain, for the several chemical tests described above. Should the first residue by evaporation of a portion of the chloroform prove not sufficiently pure and colorless, the residue of the whole, or of a portion, may be further purified by another digestion with hot concentrated sulphuric acid as before.

For a quantitative estimation an aliquot part of the entire chloroform solution is thoroughly purified, its final chloroform solution evaporated on a small tared watch-glass, washed with a few drops of ice-cold water, dried, and weighed. It must be understood, however, that an alkaloid cannot be separated from tissue substances without loss.[2] The ratio of the loss of alkaloid to the weight of tissue material may be approached by a parallel control analysis, but it is doubtful whether sufficient data can be obtained to justify an estimation of the quantity of alkaloid in the entire body in a case of death from poisoning.

Deposition of Strychnin in the Body.—Strychnin is eliminated unchanged to some extent by the perspiration, saliva, bile, and much more by the urine (Kobert). As a poison it has been recovered especially from the liver and the kidneys, and an unabsorbed remainder is generally to be found in the stomach and its contents. It has been found in the brain and in the blood. (See Cases 14 to 20.)

Detection in the Body after Prolonged Periods.—Authorities agree that strychnin long resists decomposition in the body. Haines[3] states that he extracted it in a ponderable quantity from a body that had been buried for nearly twelve months (see also Case 19).

[1] As to formation of a strychnin-sulphonic acid under this treatment see Stoecher, *Ber. d. chem. Ges.*, vol. xviii., p. 3429.

[2] See the writer's *Organic Analysis*, p. 461, or *Chem. News*, 1885, vol. liii., p. 78.

[3] Hamilton's *System of Legal Medicine*, vol. i., p. 459.

Alfred H. Allen states that he recovered strychnin from a stomach that had been kept untreated six years.[1]

Failure to Detect.—There are a considerable number of cases of death from undoubted strychnin poisoning in which competent chemists have been unable to find the alkaloid in the body. Haines[2] states that he examined the stomachs of two children who died suddenly with all the symptoms of strychnin poisoning, etc., but could not obtain any indication of the poison in the analysis of either case (see Cases 16 and 18).

BRUCIN.

A definition of this alkaloid is given under the general description of Strychnin, to which it is related in chemical constitution, botanic occurrence, and physiologic effect. Brucin ($C_{23}H_{26}N_2O_4$), a dimethoxy-strychnin, is much less stable than strychnin itself, especially in resistance to oxidizing agents, and much less potent in effect as a poison, although similar in its action. Taylor quotes Magendie as regarding brucin as having one-twelfth the strength of strychnin, but gives more credence to the ratio of one-sixth, ascribed by Andral. Reichert found that brucin is from forty to fifty times less powerful than strychnin as a convulsant, is more poisonous to the sensory nerves, and less rapidly absorbed. Brucin exerts a local anesthetic effect not obtained from strychnin. The exact determination of the potency of brucin has been embarrassed by the difficulty of absolute exclusion of strychnin. Each of these alkaloids must be separated from the other in manufacture. Therefore an article of brucin obtained from an ordinary dealer is liable to contain enough strychnin to exert a sensible effect, and the impurity is the more likely to be overlooked because the color-test for strychnin is obscured by the presence of no more than an equal weight of brucin. A method of separation of strychnin from brucin should precede the test of the latter for the presence of the former. The nux vomica seeds contain both these alkaloids; the bark contains brucin with very little strychnin, and in the bark of some species of strychnos brucin is found without strychnin. Therefore in cases of poisoning by the crude drug it is desirable to assay it for content of brucin as well as for strychnin.

The **symptoms** and the **poisonous quantities** of brucin are indicated as nearly as they can be by a comparison with those of strychnin, qualified by the statements given in the last paragraph. Several cases of death from brucin poisoning are reported.

Chemical tests for brucin are as follows: To a colorless residue to be tested, on a white porcelain surface, add a drop of concentrated sulphuric acid. With this brucin itself gives no color. A very little nitric acid is now added on the point of a sharp glass rod, when a blood-red color reveals brucin. The test may be varied by treating the residue first with dilute nitric acid, then with concentrated sulphuric acid. Treatment of the residue with nitric acid, warming

[1] *Commercial Organic Analysis*, vol. iii., pt. ii., p. 376.
[2] Hamilton's *System of Legal Medicine*, vol. i., p. 459.

gently, and then touching the spot with a solution of stannous chlorid, brings out a fine purple color as a very delicate and distinctive test for brucin (see No. 2, Plate 7).

To test for strychnin in the presence of brucin, treat the residue with concentrated sulphuric acid, then with a very little nitric acid, and after the red color has changed to yellow, add a minute fragment of potassium permanganate or of potassium dichromate. A blue or blue-violet color, the "fading purple," indicates strychnin. In this test the interference of the brucin is avoided by its decomposition or conversion into derivatives. Keller estimates strychnin in a mixture with brucin after decomposing the latter, by digesting the dry alkaloids, first with sulphuric acid of 10 per cent. strength, then in the cold with one-tenth as much nitric acid of specific gravity 1.42, leaving for one and one-half hours in the cold. In the "fading purple" test for strychnin the interference of brucin is diminished by using the permanganate as an oxidizing agent, when the brucin is decomposed to a greater extent than results from the dichromate.

In the separation of brucin from tissues and foods advantage is to be taken of the fact that brucin is much more soluble in alcohol than is strychnin. Absolute alcohol is a good separative solvent for free brucin, as is also alcohol of 50 per cent. strength.[1] Brucin has less resistance to decomposition by oxidizing influences than has strychnin, and must not be heated with concentrated sulphuric acid on the water-bath, as directed for the last-named alkaloid.

Case of Poisoning by Brucin.—A middle-aged man, after a meal, took 2 grains (0.13 gm.) of brucin, prescribed by a medical practitioner. In two hours he was found seated in a chair which he held by the arm, with dread depicted on his face, afraid to move or be touched lest he should fall into convulsions. Under treatment with an emetic, followed by chloral and hypodermic administration of morphin in doses of $\frac{3}{8}$ of a grain (0.025 gm.), the convulsive state gradually disappeared.[2]

THE VERATRUM ALKALOIDS.

The most representative of the various alkaloids of hellebore and cevadilla are the following:

1. Cevadin, the veratrin of Merck, "crystallized veratrin" ($C_{32}H_{49}NO_9$) in cevadilla (sabadilla) seeds, from the plant *Asagraea officinalis*, and an important part of medicinal veratrin of the United States Pharmacopeia. It is soluble in ether and is sternutatory.

2. Veratrin of Wright and of Couerbe ($C_{37}H_{53}NO_{11}$), "amorphous veratrin" in cevadilla seeds, in traces in *Veratrum viride*, and in veratrin of the United States Pharmacopeia. Amorphous as free base, it forms crystallizable salts. A saponifiable alkaloid, yielding another base, verin.

3. Jervin ($C_{26}H_{37}NO_3$), in *Veratrum viride* (0.02 per cent.), in *Veratrum album* (0.13 per cent.) (E. Schmidt). It is insoluble in ether.

[1] See Prescott's *Organic Analysis*, 1887, p. 458.
[2] T. S. Sozinskey, *Med. and Surg. Reporter*, Philadelphia, 1882, vol. xlvii., p. 194.

The veratrin of the United States Pharmacopeia is defined as "a mixture of alkaloids obtained from the seed of *Asagræa officinalis.*" It is especially characterized by its content of cevadin, the alkaloid first above named.

Veratrum viride, or American hellebore, contains cevadin and jervin, as above stated, and traces of veratrin. It is used in medicine in this country in the fluid extract (1 to 1) and in the tincture (1 to $2\frac{1}{2}$). The tincture of the British Pharmacopeia has twice the strength of that of the United States Pharmacopeia.

Symptoms of Poisoning by Veratrum Viride or by Veratrin (U. S. P.).—Great nausea, burning and pain in the stomach and bowels, violent vomiting, prostration, cold extremities, a small, slow, and feeble pulse, the heart action very weak, dizziness, blindness, dilatation of pupils, sleepiness, coma, sometimes stertorous breathing. There is great variation in the symptoms. With *Veratrum viride* the pulse is first reduced in force, then in frequency. It acts as a spinal and arterial depressant. The jervin acts directly upon the heart muscle and depresses the spinal motor centers (Wood). Death is due to asphyxia. In fatal cases death occurs sometimes in five or six hours, but more often after twenty-four hours or longer.

The **fatal quantity** of the drug cannot be safely declared, on account of great differences in susceptibility to this agent. The medicinal dose of the fluid extract is from 1 to 3 minims (0.06 to 0.18 c.c.) every hour or two, the results to be closely watched and the administration suspended if there is nausea (see notes of cases of poisoning below).

Treatment of Poisoning by Hellebore.—The stomach should be well washed out with warm water. The patient should then be kept strictly upon his back, with the head lower than the feet, and vomiting should be restrained. Warm applications, especially to the feet, and external stimuli should be kept up. A dose of tincture of opium may be administered by the rectum, undiluted spirits by the mouth, and ammonia at discretion. To meet failure of respiration Böhm and Lissauer (1887) propose atropin hypodermically, but strychnin and digitalis are more directly indicated.

Cases of Poisoning by Veratrum viride.

Case 1.—A man took 1 dram (3.7 c.c.) of tincture, equal to 12 grains (0.77 gm.) of the powder. The symptoms consisted of collapse, cold and perspiring skin, scarcely perceptible pulse, pain in the stomach, but no purging. Result, recovery.[1]

Case 2.—A child of eighteen months took four or five doses of 4 minims (0.24 c.c.) each, and then one dose of 16 minims (1 c.c.) of the tincture. There occurred attempts at vomiting, unconsciousness, and stertorous breathing beginning eight hours after the first dose; the pulse was slow, the skin in cold perspiration. Result, death in thirteen hours.[2]

Case 3.—A woman of fifty years took 70 minims (4.3 c.c.) of a fluid extract of veratrum viride in two doses. Pain in the stomach, nausea, and vomiting occurred in two hours after the first dose. In two hours after the second dose the symptoms became more severe and there was great prostration. After twelve

[1] Edwards, *Med. Times and Gaz.*, 1863, vol. i., p. 5.
[2] J. C. Harris, *Amer. Jour. Med. Sci.*, 1865, p. 284.

hours the stools were bloody. Vomiting continued for four weeks, when the patient died.[1]

CASE 4.—An adult male took a teaspoonful of the fluid extract of veratrum viride. In thirty minutes he became speechless, with violent vomiting, the pulse being almost imperceptible, and the skin bathed in cold perspiration. The patient recovered.[2]

The same physician reports another case of like dose with similar result, loss of speech and of locomotion being among the symptoms.

Postmortem Appearances.—These are neither distinctive nor uniform, but usually include signs of congestion of the lining of the stomach and bowels and sometimes hyperemia of the brain and its membranes. The kidneys should be examined.

Chemical Tests for Veratrum Alkaloids.—As to the molecular constitution of these bases, Guareschi expresses the opinion that they are related to the aconite alkaloids and, like the latter, are quinolin derivatives. Pyridin products are obtained in dry distillation. They are not so unstable as the alkaloids of aconite.

The distinctive color-test with concentrated sulphuric acid is given by cevadin, veratrin, and jervin, the alkaloids numbered 1, 2, and 3 at the beginning of this section, and therefore given by the total alkaloids of veratrum viride and by the veratrin of the United States Pharmacopeia. The residue of dry alkaloid, on white porcelain, is covered with an excess of pure concentrated sulphuric acid. The veratrum alkaloids dissolve to a yellow solution, which, on standing, changes to a bright red, as observed in looking through it to the white ground, gradually deepening and slightly darkening, then remaining persistent for hours. (See No. 6, Plate 7.) If the test is warmed, the changes just stated take place quite rapidly, but the deep, dark-red color remains some time. Sufficient quantities show a green fluorescence by reflected light. It is the persistence of the color that is the most characteristic of veratrum alkaloid in this test. Various organic compounds give red colors with sulphuric acid, but the analyst, in comparing the results with that of veratrum alkaloid, finds the latter to possess distinctive features.

Strong hydrochloric acid dissolves veratrin without change of color, but upon boiling, a bright-red color is developed that persists a long time. This result is strongly indicative. Fröhde's reagent gives a result sooner than sulphuric acid alone, and a distinction from some other bodies. The action of concentrated sulphuric acid with cane-sugar is characteristic. The alkaloidal residue is rubbed with six times its quantity of cane-sugar, and the powder treated with a few drops of concentrated sulphuric acid. The color product, in case of veratrum alkaloids, is yellowish, then green, and lastly a clear blue (Weppen). A reaction with furfurol is recommended by G. Laves (1892): To 16 minims (1 c.c.) pure concentrated sulphuric acid, in a test-tube, add three or four drops of a 1 per cent. solution of furfurol in water and mix. Now add from three to five drops of a water solution to be tested

[1] T. M. Johnson, *Buffalo Med. and Surg. Jour.*, 1866, p. 133.
[2] J. B. Buckingham, *Amer. Jour. Med. Sci.*, 1865, p. 563.

for veratrum alkaloids, letting this flow down the side of the tube without shaking. Veratrum alkaloids give a dark layer, changing to dark green, blue to violet blue, at contact with the acid.

Cevadin ($C_{32}H_{49}NO_9$) or "crystallized veratrin" crystallizes in transparent, colorless, concentric groups of needles, with a melting-point of 205° C. (Ernst Schmidt). It forms a gold salt, crystallizing from hot alcohol in yellow needles—$C_{32}H_{49}NO_9.HCl.AuCl_3$.

Jervin ($C_{26}H_{37}NO_3$) is especially characterized by the sparing solubility of its salts of sulphuric, hydrochloric, and nitric acids. These salts, sparingly soluble in water, are less soluble in their respective acids, and are precipitated accordingly by adding the acid in some excess to an acetic acid solution of the alkaloid. The precipitate is crystalline. The free alkaloid melts at 238° C. This alkaloid alone, in the color-test with pure sulphuric acid above given, gives a final color bearing more on the brown than the color obtained from cevadin and veratrin.

A characteristic physiologic test of veratrum alkaloids is obtained with the frog by hypodermic administration. The animal suffers immediate slowing of the heart, is apt to vomit, has spasmodic muscular contractions, and if the amount be sufficient, finally dies.

Ptomains have been found closely resembling veratrin under the color-tests, but not at all responding to the physiologic test.[1]

In separation from the tissues or contents of the stomach it must be borne in mind that chloroform, benzene, and amyl alcohol respectively dissolve veratrum alkaloids, to some extent, from *acidulous*, as well as from alkaline, water solutions. Chloroform is, in general, the best solvent, applying it to feebly alkaline solutions. The process given under Atropin may be employed, omitting the chloroformic washing of the acidulous solution, and continuing the steps of purification until a sufficiently colorless residue is obtained.

The kidneys and the urine should be subjected to analysis, as this poison is eliminated to some extent by the kidneys.

MUSHROOM POISONS.

The poisonous principles of mushrooms are, in general, to be classed with the hurtful products of other fungi. The eminent pharmacologist Kobert, in 1899, classified the known poisons of the fungi as follows:

(1) Acids, such as helvellic acid; (2) alkaloids, as muscarin and neurin; (3) albuminoids, as the enzymes and toxalbumins. Further, muscarin acts upon the nerves; certain fungi, as species of Lactarius, cause local irritation; other products, like helvellic acid and the phallin of *Amanita phalloides*, act primarily upon the blood.

W. G. Farlow, of Harvard University, has given (1898) the following statements of fleshy fungi (mushrooms) *liable to be poisonous:* (1) Those in the button or unexpanded state; also those in which the flesh has begun to decay, even if but slightly; (2) those having a stalk with a swollen base, surrounded by a sac-like envelope, especially if the gills

[1] Consult p. 704 in the section on Ptomains and Other Bacterial Products in their Relation to Toxicology.

are white; (3) those having a milky juice, unless the milk is reddish; (4) those in which the cap or pileus is thin in proportion to the gills, and in which the gills are nearly all of equal length, especially if the pileus is bright colored; (5) all tube-bearing fungi in which the flesh changes color when cut or broken, or where the mouths of the tubes are reddish; (6) generally those which have a sort of spider-web or flocculent ring around the upper part of the stalk. All the foregoing are to be avoided as poisonous or doubtful.[1]

It was reported in 1883, by Dupetit,[2] that all mushrooms are in some measure poisonous when uncooked. He found the sap, subcutaneously injected into rabbits and other animals, to cause death. The

FIG. 59.—*Amanita muscaria* (one-half natural size).

poisonous constituent was not removed by a Pasteur filter, and had the chemical properties of a soluble ferment. The spores of the mature large puff-ball are liable to act as a poison (see Case 7).

The alkaloid **muscarin** is found in certain fleshy fungi, especially the *Agaricus muscarius* (*Amanita muscaria*) and the *Agaricus pantherinus* (*Amanita pantherina*). It is also known as a ptomain or product of putrefaction.[3] Muscarin is a chemical relative of cholin, and can be

[1] The liability of mistake in the selection and use of mushrooms for food is exemplified in an account, given in 1896 in the *Progrès Médical*, that a man and his wife who had dealt in edible mushrooms in Paris for more than fifty years themselves both died at the same time from mushroom poisoning.

[2] *Compt. rend.*, vol. xcv., p. 1367.

[3] Brieger, 1886.

synthetically produced from the latter. Muscarin ($C_5H_{15}NO_3$) has the constitution $(CH_3)_3N.CH_2CH(OH)_2.OH$. Cholin ($C_5H_{15}NO_2$) has the constitution $(CH_3)_3N.CH_2CH_2(OH).OH$. Cholin is also convertible into neurin, $(CH_3)_3N.(CH:CH_2).OH$, a ptomain. Of these three quaternary ammonium hydroxids only muscarin has the constitution of an aldehyd and it is far more poisonous than cholin or neurin. Muscarin is capable of reduction to cholin and, more easily, of oxidation to betain, $(CH_3)_3N.CH_2CO_2H.OH$. Dr. D. W. Prentiss[1] states that while *Amanita muscaria* is poisonous to flies, they are not poisoned by the alkaloid muscarin, and he infers the presence of another and a volatile poisonous principle in this species.

The **symptoms of poisoning by muscarin,** or by mushrooms containing it, are slowing of the pulse, contraction of the pupils of the eyes, salivation and lacrimation, spasm of the accommodation of the eye, tetanic spasms of the stomach and intestines becoming violent, and stoppage of the heart-beats. Decided symptoms are caused by so small a quantity of muscarin as from $\frac{1}{60}$ to $\frac{1}{20}$ of a grain (1 to 3 milligrams) (Kobert). It is a quite direct antagonist of atropin (Schmiedeberg), and the latter is directly indicated in the treatment of muscarin poisoning. It has been found that the physiologic effects of the muscarin from mushrooms are identical with those of synthetic muscarin from cholin. It is largely excreted through the kidneys.

Fig. 60.—*Amanita phalloides* (death cup).

The fungus *Amanita muscaria* (*Agaricus muscarius*), or fly-blown agaric, having the size and shape of a common mushroom but the outer surface of the pileus of a bright-red or sometimes yellowish color, has been used as a fly-poison. Similar species in northeastern Asia have been used as an intoxicant. The poison is so far excreted in the urine that the latter exerts the same intoxicating effect.

In chemical tests for muscarin regard must be had to its properties. It is odorless and tasteless, freely soluble in water and in alcohol, slightly soluble in chloroform and still less in ether, usually obtained as a syrupy liquid, but crystallizing when anhydrous. It has a very strong alkaline reaction, and, in accord with its quaternary constitution, resembles a metallic alkali rather than a volatile alkali. It melts and darkens at 80° C., and above 100° C. becomes solid again (Ernst Schmidt). At higher temperature it yields a slight vapor of tobacco-like odor. With excess of platinum chlorid, evaporating in vacuum, a platinum chlorid of muscarin crystallizes in small octahedra. The corresponding double salt of cholin crystallizes in plates. In order to recover free muscarin from its platinum chlorid add an excess of

[1] *Phila. Med. Jour.*, 1898, vol. ii., p. 607.

potassium chlorid, evaporate to dryness, extract with strong alcohol, evaporate by gentle warmth to dryness, and extract again with strong alcohol. The residue is muscarin chlorid. By treatment with moist silver oxid and water the free muscarin can be obtained. In postmortem analysis for muscarin the urine should be examined for it.

The *Amanita phalloides*, or death-cup, and other species of Amanita are said to be the most frequently occurring of the poisonous mushrooms liable to be eaten in this country. These belong to Kobert's third class, containing *toxalbumins*.

Phallin, the toxalbumin of *Amanita phalloides*, acts mainly upon the blood. It is said to be absorbed from the intestines instead of the stomach, and therefore its poisonous effect is later than that of muscarin. Among the *symptoms* of poisoning by these are great prostration, cold sweat, stupor, collapse, sometimes fever and rapid pulse. The pupils are generally dilated—in distinction from poisoning by muscarin-bearing fungi. The blood is seriously affected, and after death it remains fluid and stains the tissues. The mucous membrane is irritated and often inflamed. The *treatment* consists in the prompt evacuation and washing out of the stomach, prompt catharsis, and the relief of symptoms as they arise. Norris[1] advises, in the stage of collapse, the intravenous injection of a sterilized warm solution of common salt (7 : 1000).

Helvellic acid ($C_{12}H_{20}O_7$) is the chief poisonous principle in *Helvella esculenta* and other species of this genus. Cholin has been found in *Helvella esculenta*, but this is but a feebly poisonous base, its fatal dose for rabbits being 7⅔ grains (0.5 gm.) per kilogram of body weight. The poisonous acids of these fungi are dissipated or destroyed in the drying of the plant, also in continued boiling. The poisonous effects are exerted primarily upon the blood, as stated by Kobert.

Cases of Poisoning by Mushrooms.

Case 1.—Four soldiers ate of *Amanita muscaria*. Within six hours all were taken with griping pains, thirst, profuse purging, with swollen abdomen, a sense of suffocation, and delirium. Death was the result with the four. Others who partook at the same time recovered, some of them after prolonged coma.[2]

Case 2.—In Winnetka, Ill., a woman of fifty-five years ate a third of a raw mushroom (*Amanita phalloides*) at 10 a. m. At 7 p. m., after supper, symptoms of poisoning began with cramps in the abdomen, increasing, and at last affecting the thighs, with vomiting at 8 p. m. Patient was then found in collapse by physician, with a temperature of 100.5° F. The pulse was rapid, the respirations were shallow, there were moderate tympanites and slight tenderness over the entire abdomen, the pupils were contracted, there were cold perspiration, intense retching, profuse watery stools, and mild delirium. Under treatment with morphin, atropin, strychnin, stimulants, and antacids the symptoms at midnight had gradually abated and the patient recovered. The opinion of the physician that the poisoning was by *Amanita phalloides* seems to have been based in good part upon the delay in the symptoms, due to the statement that phallin is absorbed by the small intestine instead of by the stomach.[3]

Case 3.—Count de V., residing in Washington, D. C., ate about two dozen mushrooms at breakfast. With him Dr. K. ate about one dozen of the same.

[1] *Phila. Med. Jour.*, 1898, vol. ii., p. 810.
[2] Corvisart, *Jour. de Med.*, vol. xxxi., p. 323.
[3] H. C. R. Norris, *Phila. Med. Jour.*, 1899, vol. ii., p. 810.

The mushrooms had been collected the previous day by direction of the Count, who thought he recognized them as of an edible species—*Amanita Cæsaria*. In the case of the Count, symptoms of poisoning began in a quarter of an hour, with collapse, blindness, unconsciousness, and convulsions. Emetics and hypodermic administration of apomorphin and atropin were employed without avail, the patient dying on the evening of the second day. Dr. K. suffered like symptoms, beginning later. Vomiting was not obtained until evening. There was no rise in temperature. He remained in the hospital a week before recovery was complete.[1]

CASE 4.—A man, a woman, and a child ate *Amanita muscaria* (fly-mushroom) and *Amanita phalloides* (death-cup) at Chambersburg, Pa. The man ate about a dozen, the woman about eight, and the child (five years old) four or five at midday dinner, and a few more were eaten at supper. They all became ill at 10 P. M. and were very sick at midnight. The symptoms included unconsciousness and convulsions. On the third day the woman and the child died. The man gradually recovered.[2]

CASE 5.—An adult male and a boy of thirteen years both ate of *Agaricus procerus*. In half an hour there was extreme giddiness, and in an hour both became insensible. After the use of the stomach-pump and the action of stimulants sensibility returned, with convulsive spasms and delirium. The bowels were not affected, nor was there pain. The pupils were contracted only during the sleep. Both patients recovered.[3]

CASE 6.—The *Amanita pantherina* was eaten by a man and a little girl of five years. In two and a half hours there were faintness, nausea, and delirium; in eleven hours abdominal tenderness and stupor. The pupils of the child were contracted. Both recovered.[4]

CASE 7.—In Edinburgh Mr. Sadler, when about to deliver a lecture upon edible and poisonous fungi, accidentally swallowed some of the spores of a large species of puff-ball (*Lycoperdon giganteum*). In an hour and a half he became very ill, suffering violent pain in the abdomen. The pain did not subside until after nine days. (When young, the puff-ball is edible.)[5]

CASE 8.—A woman and a child ate of *Amanita citrina* (yellow color) fried. After seven hours the child became feverish and thirsty, with severe pain in the stomach and a feeling of constriction in the throat. The pupils were dilated. Convulsions and insensibility supervened, and the child died forty-one hours after eating the fungi. The woman had like symptoms, partially recovered, had a collapse, and died on the fifth day.[6]

CASE 9.—In London, in 1873, a man was tried in the Central Criminal Court for poisoning a young woman by giving her mushrooms. She had fried them herself, eaten them for breakfast, and died on the evening of the same day. It was alleged that the accused had a motive for the act charged. In absence of proof that he knew the fungi to be poisonous he was acquitted.[7]

[1] D. W. Prentiss, *Phila. Med. Jour.*, 1898, vol. ii., p. 607 (with a full report upon "Mushroom Poisoning," and with account likewise of Case 4).

[2] Dr. Prentiss, as quoted above.

[3] Peddie, *Edinburgh Med. and Surg. Jour.*, vol. xlix., p. 192.

[4] Husemann, *Jahresbericht*, 1872, p. 534.

[5] *Brit. Med. Jour.*, 1874, vol. i., p. 595.

[6] Alfred Swaine Taylor, *Guy's Hosp. Reps.*, October, 1865, p. 382.

[7] *Lancet*, London, October 4, 1873, p. 501.

NON-ALKALOIDAL ORGANIC POISONS.

PETROLEUM.

PETROLEUM consists of a number of hydrocarbons, belonging, for the most part, to the paraffin series. In crude petroleum there are gaseous, liquid, and solid hydrocarbons, together with many impurities. By distillation crude petroleum is divided into a number of fractions to which various commercial names have been given. The fractions with a low boiling-point are known as petroleum ether, ligroin, gasolin, naphtha, benzin, etc. The fraction boiling at 150° C. to about 300° C. (302° to 572° F.) is ordinary kerosene, while that which is liquid at ordinary temperatures but which boils at a temperature above 250° to 300° C. (482° to 572° F.) is "paraffin oil" or "mineral oil." When the substances named are distilled off, vaselin and paraffin are left. All these preparations are somewhat poisonous; in some cases the vapor, in others the liquid or even the solids, have caused poisoning. Crude petroleum contains sulphur compounds that greatly increase the toxicity of both the vapors and the liquid itself. Cases of poisoning by the vapors of petroleum are, for the most part, "industrial" or accidental; they occur most frequently among persons engaged in industries requiring the use of petroleum or some of its products, as in petroleum refineries and in those industries in which benzin, naphtha, etc., are used as solvents—in rubber factories, for example.[1] In most of these cases some of the symptoms are doubtlessly due in part to the contact of the poison with the body or to small quantities accidentally taken internally.

The **symptoms** are numerous and varied, and are due partly to the local action and partly to an effect upon the central nervous system. Irritation of the mucous membranes may lead to cough and to chronic inflammation of the respiratory organs. Various forms of skin eruptions[2] and boils are described. The general symptoms observed among workmen in petroleum refineries, etc., are weakness, hallucinations, and palpitation of the heart. In more acute cases[3] the patient becomes very pale, the lips are livid, the respiration is slow, and the heart's action is weak and scarcely perceptible; insensibility, convulsions, and death may follow unless the person is immediately taken into the open air. Often a condition similar to that of drunkenness ("naphtha-drunk") results.

Many cases of poisoning in which the liquid was swallowed have been reported; most of these cases were accidental, occurring especially among

[1] Chittenden and Farlow, *Boston Med. and Surg. Jour.*, vol. cxxvi., pp. 621, 640.

[2] Lewin, *Virchow's Archiv*, vol. cxii., p. 35; Mitchel, *Med. News*, vol. liii., p. 152; *cf.* Sharp, *ibid.*, p. 150.

[3] Foulerton, *Lancet*, 1886, vol. ii., p. 865.

children, whereas a few were due to attempts at suicide or murder. Fatal results have rarely been reported. Two groups of symptoms have been described—those due to the local action of the poison upon the stomach and those due to the action upon the central nervous system. The former symptoms are especially marked in those cases in which products having a high boiling-point (paraffin oil, kerosene, etc.) have been swallowed; the nervous symptoms have been especially marked after those products having a low boiling-point (benzin, naphtha, etc.).

The **gastric symptoms**[1] consist in a burning pain in the throat and stomach, vomiting, colicky pains, diarrhea, great thirst, painful micturition, etc. The odor of petroleum is on the breath. The statement is frequently made that in such cases the urine contains petroleum, but this seems to be incorrect;[2] it has often, however, the odor of violets.

Several cases of poisoning have been reported recently[3] in Germany from the use of paraffin oil or vaselin in the making of bread. The symptoms were catarrh of the stomach, as well as general nervous symptoms. The internal use of vaselin in the treatment of colds has given rise to similar symptoms.[4]

The **general symptoms** of petroleum poisoning[5] consist in headache, dizziness, accelerated heart action, labored respiration, collapse, stupor, unconsciousness, and, more rarely, convulsions; sometimes there is a rise of temperature. If the pain from the irritation of the stomach is not too great, drowsiness[6] is a prominent symptom. Unconsciousness has come on in from ten to fifteen minutes after the swallowing of less than half an ounce of benzin.

Fatal Dose and Period.—Deaths from petroleum and its products have been so rare that the fatal dose is not known. Death is said to have followed in seventeen hours the taking of about half an ounce of benzin. On the other hand, a pint of petroleum, taken by a woman with suicidal intent, did not cause death. A teaspoonful of paraffin oil caused severe, but not fatal, symptoms in a child of two years. Another child died in ten minutes after taking a "swallow" of benzin;[7] in another case a child two years old died a few hours after taking an unknown quantity of petroleum.[8] A case is also recorded in which a man died a few hours after drinking an unknown quantity of paraffin oil.

The **treatment** consists in washing out the stomach or administering emetics and the use of purgatives. In cases of collapse, warm baths with cold affusions are recommended; the usual respiratory and cardiac stimulants are also indicated.

[1] Vincent, *Brit. Med. Jour.*, 1886, vol. i., p. 543; Friedeberg, *Centralbl. f. innere Med.*, 1902, p. 1041. [2] Lewin, *loc. cit.*

[3] Dunbar, *Deutsch. med. Wochenschr.*, 1896, vol. xxii., p. 33.

[4] Robinson, *Brit. Med. Jour.*, 1886, vol. i., p. 296.

[5] Witthauer, *Munch. med. Wochenschr.*, 1896, vol. xliii., p. 915; Rosenthal, *Centralbl. f. innere Med.*, 1894, vol. xv., p. 281.

[6] Conrads, *Berlin. klin. Wochenschr.*, 1896, vol. xxxiii., p. 982; see also *Med. News*, vol. xlvii., p. 658.

[7] Falk, *Vierteljahrsschr. f. ger. Med.*, 1892, vol. iii., series 3, p. 399.

[8] Johannessen, *Berlin. klin. Wochenschr.*, 1896, vol. xxxiii., pp. 317, 349.

The **postmortem appearances** show nothing characteristic, but the esophagus, stomach, and small intestine may have the odor of petroleum.[1] The odor of benzin was noticed in one case after putrefaction had begun.[2]

Detection.—The constituents of petroleum may be shown in the tissues by fractional distillation, the various fractions being recognized by their odor, boiling-point, inflammability, and other properties.

METHYL-ALCOHOL.

Methyl-alcohol (methylic or wood-alcohol, pyroligneous or wood-spirit, wood-naphtha, CH_3OH) is obtained by the destructive distillation of wood. It occurs in commerce in varying degrees of purity. The crude wood-alcohol contains much acetone, some aldehyd, and similar bodies that give it an extremely disagreeable odor and taste. Purified preparations of methyl-alcohol are now extensively used under the names of "Columbian spirits" and "Colonial spirits."

Pure methyl-alcohol is a colorless liquid with a specific gravity of 0.8021 at 15.5° C. (59.5° F.). It boils at 66° C. (150.8° F.), and has a pungent taste and an aromatic odor, not very different from that of ethyl-alcohol. Methyl-alcohol is used very extensively in the arts as a substitute for ethyl- or grain-alcohol as a solvent, especially of varnishes; it has also been used, with most disastrous results to those taking the preparations, as a solvent in the preparation of essences of Jamaica ginger, peppermint, cinnamon, lemon,[3] and of spirits of cologne and bay-rum. It is also sold extensively for use in spirit-lamps.

Poisoning with methyl-alcohol is undoubtedly very common, but very few cases are reported in medical literature; in fact, almost no such cases were reported until recently, when the attention of the medical profession was directed to methyl-alcohol as a cause of blindness.

Apparently all the cases of wood-alcohol poisoning so far reported have been due to accident; most of them have been caused by the drinking of wood-spirits as a substitute for ethyl-alcohol. In a number of cases death or blindness has resulted from the use of extracts of flavoring agents that were made with methyl-alcohol; such cases have been reported especially from certain "prohibition" counties of Virginia and Maryland and from the Indian Territory[4] and Maine. Occasionally painters and varnishers are poisoned by the vapors of methyl-alcohol.[5]

Experiments on animals[6] have shown that the physiologic action of

[1] Johannessen, *loc. cit.* [2] Falk, *loc. cit.*

[3] A number of such preparations were examined for methyl-alcohol by Hynson and Dunning: see *Ophthalmic Record*, 1901, vol. x., p. 83.

[4] See Moulton, *Jour. Amer. Med. Assoc.*, 1901, vol. xxxvii., p. 1447.

[5] Patillo, *Ophthalmic Record*, 1899, vol. viii., p. 599; Hale, *Jour. Amer. Med. Assoc.*, 1901, vol. xxxvii., p. 1450; De Schweinitz, *Ophthalmic Record*, 1901, vol. x., p. 289.

[6] Pohl, *Arch. f. exper. Path. u. Pharmakol.*, vol. xxxi., p. 281; Joffroy and Serveaux, *Arch. de Méd. exp.*, 1896, vol. viii., p. 473; Hunt, *Johns Hopkins Hospital Bulletin*, vol. xiii., p. 213.

methyl-alcohol differs in some respects from that of ethyl-alcohol. Thus, according to recent experiments, a slightly larger single dose of methyl-alcohol is necessary to cause death; the effects of the methyl-alcohol, however, are longer continued, so that the animal succumbs very much more quickly to small repeated doses of methyl- than to corresponding doses of ethyl-alcohol. The narcosis caused by methyl-alcohol is usually followed by a comatose condition continuing for days. Methyl-alcohol also differs from ethyl-alcohol in being but partially oxidized in the body; some of it appears in the urine in the form of formates.[1] As it has been shown that sodium formate[2] is about eight times more toxic than methyl-alcohol, we have an interesting illustration of the fact that the body sometimes converts one poison into another of much greater strength. The formate formed from methyl-alcohol is excreted very slowly—in the course of several days; this slow excretion, which was shown by Pohl to depend upon a very slow formation of formic acid in the tissues, probably explains the prolonged effect of a single dose of methyl-alcohol and also the fact that methyl-alcohol is so toxic in small repeated doses. Similar differences in action have been observed in cases of poisoning in man.

Symptoms.—As comparatively few cases of wood-alcohol poisoning have been reported, it is not possible to give many details concerning the general symptoms. In some cases there has been a condition of exhilaration and excitement closely similar to that caused by ethyl-alcohol.[3] In nearly all cases there have been severe headache, nausea, violent and long-continued vomiting, profuse sweating, dilatation of the pupils, delirium, and finally coma that continued for days; all these symptoms are much more marked after taking methyl-alcohol than after taking ethyl-alcohol. In two cases reported by Kuhnt[4] there were no marked symptoms for about twenty-four hours, and the patients continued their work; the first symptoms were feelings of chilliness and vertigo, which were followed by deep, long-continued sleep, and in one case by death.

In a case of Dr. Ellett's, cited by Moulton,[5] the patient, who was already on a spree, became comatose within three hours after drinking half a pint of wood-alcohol. He continued in this condition for the greater part of four days, but recovered except for the onset of partial blindness.

The symptom that has aroused most interest is the partial or total blindness that has occurred in a large percentage of severe cases of poisoning by methyl-alcohol. Thomson,[6] Woods,[7] Harlan,[8] and others have reported a number of cases resulting from the use of the essence

[1] Pohl, *loc. cit.*
[2] See Mayer, *Arch. f. exper. Path. u. Pharmakol.*, vol. xxi., p. 122.
[3] MacCoy and Michael, *Med. Record*, 1898, vol. liii., p. 777.
[4] Kuhnt, *Zeitschr. f. Augenheilk.*, 1899, vol. i., p. 40.
[5] Moulton, *loc. cit.*
[6] Thomson, *Ophthalmic Record*, 1897, vol. vi., p. 579.
[7] Woods, *ibid.*, 1899, vol. viii., p. 55.
[8] Harlan, *ibid.*, 1901, vol. x., p. 81; see also Wurdamann, *Amer. Med.*, vol. ii., p. 995, and Burnett, *Therap. Gaz.*, 1901, p. 801.

of Jamaica ginger, etc., made with methyl-alcohol,[1] and Moulton[2] and others cases caused by the use of pure methyl-alcohol itself. Moulton[3] cites 15 cases in which blindness was caused by methyl-alcohol, and 12 cases due to the use of Jamaica ginger, etc. The symptoms have been very uniform in all cases. As a rule, no unusual symptoms were noticed for a day or two or even longer[4] after the poison was taken. Then there was a feeling of pain about the eyes. The vision became blurred, and in from twelve to forty-eight hours the patient became totally blind; after a few days there was a gradual return of sight—to some, useful vision, only to be lost again in a short time or within a few weeks. The blindness is due to an atrophy of the optic nerve.[5] In nearly every case a central scotoma was demonstrated. In some cases some peripheral vision was preserved, but the field was always narrowed. Sometimes the color sense is chiefly affected.[6]

In some cases of methyl-alcohol poisoning practically the only symptom has been the blindness. Thus Gifford[7] reports a case in which a man became temporarily blind in three days from drinking cologne-spirits made with methyl-alcohol; so slight were the other symptoms that the patient had forgotten that he had drunk the cologne. In another case in which a large quantity of the poison had been taken the patient was comatose for two or three days and then awoke almost blind.[8]

Fatal Dose.—Moulton[9] mentions an instance in which five men drank about half a pint of wood-alcohol each; all became very ill; two died within twenty-four hours, two recovered entirely, while the fifth recovered, but with complete atrophy of the optic nerve and retina of the right eye and great contraction of the field of vision of the left eye. In other cases[10] considerably less than half a pint of wood-alcohol caused death. Complete nerve atrophy is said to have resulted from the taking of from two to five teaspoonfuls of methyl-alcohol.[11]

Fatal Period.—In some cases, when the dose was very large or was taken upon an empty stomach, death occurred in a few hours, but, as a rule, it has not occurred for one[12] or two days.[13]

[1] In some of the earlier cases of poisoning by these essences methyl-alcohol was not recognized as the toxic agent; later studies, however, admit of no doubt that such was the case.

[2] Moulton, *Therap. Gaz.*, 1899, vol. viii., p. 335.

[3] Moulton, *Jour. Amer. Med. Assoc.*, 1901, vol. xxxvii., p. 1448.

[4] In a case reported by Stieren (*Jour. Amer. Med. Assoc.*, vol. xxxvi., p. 34) complete blindness apparently came on within about three hours after the patient had consumed 12 ounces (355 c.c.) of essence of Jamaica ginger; he had been drinking heavily the day before. With prompt and energetic treatment there was great improvement in vision.

[5] The exact pathogenesis of methyl-alcohol amblyopia is receiving considerable attention at present; see Friedenwald, *Ophthalmic Record*, 1901, p. 429, and Birch-Hirschfeld, *von Graefe's Arch. f. Ophthal.*, 1902, vol. liv., p. 68. The latter author obtained blindness in monkeys from small repeated doses of pure methyl-alcohol.

[6] Burnett, *Ophthalmic Record*, vol. xi., p. 309.

[7] Gifford, *ibid.*, 1901, vol. x., p. 342.

[8] Harlan, *loc. cit.*

[9] Moulton, *Ophthalmic Record*, 1899, vol. viii., p. 335.

[10] Gifford, *ibid.*, vol. viii., p. 448 (case of Dr. Foster).

[11] Rauh, *ibid.*, p. 619.

[12] Moulton, *loc. cit.*; Gifford, *loc. cit.*

[13] Kuhnt, *loc. cit.*; Gifford, *loc. cit.*

Treatment.—In cases of poisoning by methyl-alcohol the stomach should be washed out. Stimulants, such as caffein, and digitalis, seem to be indicated. In the state of coma warm baths followed by cold affusions would probably be found useful. Some good might be expected from the administration of diuretics to hasten the excretion of the formates formed from the methyl-alcohol. The eye symptoms have apparently been treated with some success by the use of pilocarpin, potassium iodid, strychnin, and cathartics.[1]

Detection.—Methyl-alcohol can be isolated from the tissues by the method of distillation in steam (see p. 542, under Ethyl-alcohol). A few cubic centimeters of the distillate are warmed with a mixture of bichromate of potassium and sulphuric acid for the conversion of the methyl-alcohol into formic acid, and after neutralizing with calcium carbonate and precipitation of the reagents with lead acetate, the filtered liquid may be tested for formic acid. This is best done by warming the fluid with a solution of silver nitrate in ammonia. In case formic acid is present, a mirror of silver will be deposited.

ETHYL-ALCOHOL.

From almost every standpoint ethyl-alcohol must be regarded as the most important poison with which medical men and jurists have to deal; no other poison causes so many deaths or leads to or intensifies so many diseases, both physical and mental, as does alcohol in the various forms in which it is taken. Yet the number of deaths due to acute alcoholism, with which we are especially concerned here, is comparatively small when the enormous number of cases of intoxication is taken into consideration. Baudy[2] reports 1129 hospital cases of alcoholism, acute and chronic, in which 14 died—a mortality of 1.5 per cent.; as many of these were chronic cases, in which the mortality is greater than in the acute, the death-rate in the latter is still smaller. Other hospital statistics show a higher death-rate,—from 5 to 10 per cent.,—but the number of acute cases is not given.

Properties.—Pure ethyl-alcohol (*alcohol ethylicum, spiritus vini, alcohol* (U. S. P.), C_2H_5OH) is a "transparent, colorless, mobile, and volatile liquid, of a characteristic, rather agreeable odor, and a burning taste. Very hygroscopic. Specific gravity not higher than 0.797 at 15° C. (59° F.)."[3] It boils at 78.4° C. (173.1° F.), and is congealed at —130.5° C. (—202.9° F.). It burns with a non-luminous flame. The alcohol of commerce always contains some water (usually about nine parts by weight) and various impurities. The different alcoholic beverages vary widely in the percentage of alcohol they contain: beers have from 2 to 6 per cent.; light wines, from 10 to 12 per cent.; strong wines, such as port and sherry, from 19 to 25 per cent.; spirits, such as whisky, brandy, rum, and gin, have from 45 to 60 per cent. of alcohol

[1] See Harlan, Stieren, and Gifford.
[2] *Quarterly Journal of Inebriety*, 1900, vol. xxii., p. 58.
[3] United States Pharmacopeia.

by weight. It is the latter class of beverages (the spirits) that is responsible for most cases of acute poisoning with alcohol. These beverages, which contain in addition to ethyl-alcohol higher alcohols of the same series, such as amyl-alcohol or "fusel oil," and certain little-known bodies called "œnanthic ethers," are, as a rule, slightly more poisonous than pure alcohol diluted to the same extent with water;[1] there is no justification, however, for the view, sometimes held, that the toxicity of these spirits is due more to the impurities than to the ethyl-alcohol. On the other hand, some of the cheap grades of whisky are less poisonous than some of the more expensive ones, owing to their containing a smaller percentage of alcohol.

Physiologic Action.—Locally, alcohol is an irritant, producing redness and irritation of the skin, especially if the vapor be confined, and a burning, unpleasant taste when swallowed. With large quantities the irritation of the stomach causes nausea and vomiting, and if the use is long-continued, various pathologic changes result. Alcohol abstracts water from the tissues and precipitates proteids. The views as to the action of alcohol after its absorption into the blood have undergone considerable change within the last few years. Formerly it was thought to be primarily a stimulant to the vascular, respiratory, and central nervous systems; now many eminent pharmacologists hold that it is at all times a depressant of these functions, and that the symptoms of stimulation are in reality due to a paralysis of inhibitory functions. All are agreed that in large doses the action of alcohol is purely that of a depressant. Alcohol fulfils the theoretic requirements of a "food," but in only very exceptional, if, indeed, in any, cases is it to be regarded as a practicable food.[2] After the administration of a moderate quantity of alcohol it is nearly all (usually much more than 90 per cent.) oxidized in the body to carbon dioxid and water; the remainder is excreted largely by the kidneys and lungs. Opinions differ as to the extent to which alcohol passes into the milk. Some authors[3] state that only after the ingestion of very large quantities is alcohol to be found in the milk, and then in but small quantity, while others have found that after the administration of even moderate amounts of alcoholic drinks there is as much alcohol in the mother's milk as in her blood.[4] Alcohol passes freely through the placenta into the fetal circulation.[5]

Two forms of poisoning with alcohol are recognized—acute and chronic. Certain acute symptoms occurring in chronic alcoholism are known as delirium tremens.

Acute poisoning with alcohol is frequently the result of a foolhardy attempt, often on a wager, to drink, within a certain short period, a large quantity of some distilled liquor; in this country the beverage is usually whisky. In rare cases alcohol has been used for the

[1] See, *e. g.*, Baer, *Archiv f. (Anat. u.) Physiol.*, 1898, p. 295.

[2] For an excellent summary of this entire subject see Rosemann, *Archiv f. d. ges. Physiol.*, 1901, vol. lxxxvi., p. 449.

[3] Rosemann, *ibid.*, vol. lxxviii., p. 466.

[4] Nicloux, *Compt. rend. de la Soc. de Biol.*, December 22, 1899; and *Compt. rend. l'Acad. de Sci.*, vol. cxxx., p. 855.

[5] Nicloux, *loc. cit.*

purpose of committing murder (children[1]) or suicide; children are sometimes poisoned by it accidentally, and clinicians have reported cases[2] in which sucklings have been poisoned by the nurse taking wine or beer. It has been suggested that in the latter cases the symptoms were due to fusel oil or other by-products. Poisoning has occasionally resulted from inhaling the vapors of alcohol.

The **symptoms** of acute alcoholic intoxication depend upon a number of factors, such as the quantity and strength of the beverage taken, the rapidity with which it is taken, and the degree of tolerance which the person has established. In most cases of intoxication the first symptoms are a flushing of the face and hands and a feeling of warmth; then comes the stage of excitement, the exact features of which are determined largely by the individual characteristics—some persons becoming sentimental, others angry, whereas others pass into a condition of stupor. Nausea, vomiting, dizziness, with a staggering gait and incoherent speech, and other symptoms of incoördination follow. The victim finally sinks into a deep, torpid sleep from which he may usually be aroused to some extent; the pupils are dilated and the muscles of the body relaxed. The breath smells strongly of alcohol. In most cases recovery takes place within a few hours, but headache and nausea may persist for several days. After the ingestion of a very large quantity of alcohol the stage of excitement may be very brief or altogether absent; the patient falls into a deep comatose sleep; the face becomes pale or cyanotic and cold, the eyes injected and staring, the pupils dilated and scarcely reacting to light, the respiration slow, sometimes stertorous, the pulse first rapid, then slow and small. Involuntary evacuations of the fæces and urine may occur. If the patient lives for a few hours, a marked fall of the body-temperature occurs, especially if he has been exposed to cold; such temperatures as 83° or 86° F. have been repeatedly reported, while in one case the rectal temperature sank to 75° F. The immediate cause of death is usually failure of the respiration. Convulsions frequently occur in children, rarely in adults.[3] Death frequently results not from the poison, but from the victim meeting with some accident due to the loss of consciousness: he may fall out of a window or into water, or food may be forced into the trachea during vomiting. The patient may die later of pneumonia contracted during the intoxication. Many cases of sudden death occur in intemperate persons as a result of a slight excess in alcohol: in some cases the exact cause of death is not clear.[4] Long-continued illness may follow a single excess in alcohol.

Acute alcoholic insanity[5] or melancholia may develop in the course of an acute intoxication. In the former the person becomes maniacal and often commits deeds of violence, such as homicide, or he may vent his wrath on articles of furniture or on animals; in alcoholic melancholia there is often a tendency to suicide.

[1] Maschka, *Handbuch d. gericht. Med.*, vol. ii., p. 384; abstracted by Blyth, *Poisons*, p. 137.
[2] Vallin, *Bull. de l'Acad. de Med.*, 1896, vol. xxxvi., p. 442. [3] See Cases 5 and 6.
[4] Hurd, *Boston Med. and Surg. Jour.*, 1897, vol. cxxxvii., p. 619.
[5] See Vol. I., p. 638.

The **diagnosis** is usually easy, but opium poisoning, apoplexy, and uremic coma have repeatedly been mistaken for acute alcoholism.

Fatal Dose.—The fatal dose of alcohol varies with the individual and his state of health, and with the strength of the beverage and the way in which it is taken. Some of the alcohol is usually returned in the vomit. Probably in the greater number of the fatal cases reported in the adult from one to two pints of whisky [1] or brandy were taken; this would correspond to approximately 8 to 16 ounces (236 to 473 c.c.) of pure alcohol. In most cases, however, a smaller quantity would probably be fatal. From 3½ to 7 ounces (100 to 200 c.c.) of alcohol is usually considered the minimal fatal dose if taken in a concentrated form and at one time. Infants have died from two tablespoonfuls of brandy, and from 1 to 2 ounces (30 to 59 c.c.) of absolute alcohol (corresponding to from 2 to 4 ounces—59 to 118 c.c.—of whisky) would probably be fatal to most children under twelve years; yet one case is reported in which a child of three and a half years ultimately recovered after taking 12 ounces (355 c.c.) of whisky.[2]

Fatal Period.—The fatal period is variable, but death seems to have occurred in most cases in from six to ten hours after the beginning of the coma; it has occurred, however, within a few minutes, or been delayed for five or six days. Recovery is rare if the coma lasts longer than from ten to twelve hours. A low external temperature hastens the end.

Treatment.[3]—If vomiting has not taken place freely, this should be encouraged by the use of emetics or the stomach should be washed out with warm water. If collapse has occurred, the limbs should be rubbed and hot applications be made to the body. Care should be taken to maintain the body-temperature. Strong coffee, capsicum, or aromatic spirits of ammonia are recommended internally or strychnin and digitalis hypodermically. Sleep or quiet may be induced by the bromids; opium should, as a rule, be avoided. In deep coma warm baths followed by cold affusions are useful; in extreme cases artificial respiration may be necessary. In alcoholic mania morphin and hyoscin are recommended.

Postmortem Appearances.[4]—In some cases of death due to acute alcoholism the stomach and other organs appear quite normal, but, as a rule, the stomach and esophagus are of a deep-red color. There are often punctiform ecchymoses in the gastric mucous membrane, which may also be easily separated. The contents of the skull are usually markedly hyperemic; there is frequently extravasation of blood in the brain and its membranes, and edema of the brain-substance or of the membranes. There may be a serous effusion in the ventricles of the brain. Edema of the lungs is frequent. If death followed soon after the ingestion of the poison, the body may resist decomposition for an unusual length of time; cadaveric rigidity may last unusually long. The contents of the stomach and the various organs, especially the

[1] See Case 1.
[2] See Case 6.
[3] See *Medical Record*, 1900, vol. lviii., p. 1024.
[4] See Case 1.

brain, may have a well-marked odor of alcohol; if the weather is cold, this may last for a long time.

In chronic alcoholism the lesions may be marked or may be absent. Sometimes the dura mater is thick and closely adherent to the skull. The brain may be edematous, atrophied, or normal. The stomach and kidneys may show the lesions of chronic inflammation; the liver is frequently cirrhotic and fatty; the large vessels are usually atheromatous. Marked histologic changes occur in the cells of the brain and spinal cord.[1]

The subject of **chronic alcoholism** is best treated in works on general medicine, but a few words may be said about delirium tremens. Delirium tremens has been aptly characterized as only an incident in chronic alcoholism; it is especially common among those addicted to the use of distilled liquors. The fusel oil contained in the latter is held by some to contribute largely to the predisposition to delirium tremens. The attack frequently breaks out without any warning, but often it is precipitated by a temporary excess in alcohol, by an accident, by a surgical operation, or by an acute inflammation, particularly pneumonia. The attack usually begins with restlessness, sleeplessness, and tremors of the hands; then follow, in a day or two, the characteristic symptoms: hallucinations of sight, more rarely of sound, incessant movement, and incoherent muttering. The patients are anxious and frequently desire to go out, and constant watching is necessary to prevent them from jumping from windows or committing suicide in other ways. The hallucinations and illusions sometimes lead to homicidal attempts. The symptoms may subside in three or four days, or they may continue and the patient die from failure of the heart.

Sometimes a person who has suffered from delirium tremens, especially if he be an epileptic, enters into a condition of trance and may do things of which, later, he has no recollection.[2]

Cases of Poisoning by Alcohol.

Case 1.—A moderate drinker, aged twenty-five, in apparent good health, wagered that he could drink a pint of whisky within ten minutes. He drank one and a half pints of cheap whisky and started for home. He soon became unconscious, vomited, and became comatose. His face was livid, he breathed heavily, and after four or five convulsions he died in six hours from the time of drinking the spirits. Autopsy, performed thirty-six hours after death, revealed the following: Pupils widely dilated and unequal; sinuses and pia engorged; whole brain edematous; lungs engorged and lower portions dotted with pleural ecchymoses. From the stomach 14.5 c.c. (one-half ounce) of absolute alcohol were recovered; the liver contained 3.5 c.c. (55 min.).[3]

Case 2.—Laborer, aged thirty-three, drank between 10 and 15 ounces (295 to 444 c.c.) of whisky. He became intoxicated in twenty minutes, and fell to the ground in a deep sleep soon afterward. It was impossible to rouse him. The pupils were contracted to almost pin-point size (very unusual) until death took place nineteen hours later. Autopsy, thirty hours after death, showed: Rigor mortis complete; dura mater congested; no effusions in ventricles of brain.[4]

[1] Larkin and Jelliffe, *Medical Record*, 1899, vol. lvi., p. 87.

[2] See Francotte, *Journal de Neurologie et d'Hypnologie*, 1897, vol. ii., p. 24; also numerous papers by Dr. Crothers, of Hartford.

[3] Baker, *Quarterly Journal of Inebriety*, 1900, vol. xii., p. 105.

[4] Berry, *Lancet*, 1898, vol. i., p. 723.

CASE 3.—Woman, aged forty-one, a periodic inebriate, drank one and one-half pints of exceptionally strong whisky. Found lying on back insensible in a few minutes; died five and a quarter hours later, without recovering consciousness. Pupils were dilated. Temperature fell to 7° F. below normal. At autopsy patches of mucous membrane of stomach were found semidetached; parts of walls were fiery red.[1]

CASE 4.—Girl, aged four, took 2 ounces (59 c.c.) of whisky. Pulmonary edema developed in eleven hours and led to death eleven hours later.[2]

CASE 5.—Girl, aged five, took quantity of whisky supposed to be but "2 dessertspoonfuls." Soon became insensible, and the use of artificial respiration became necessary at one time. After nine hours violent tetanic convulsions developed; these were relieved by chloral. Then came dyspnea, cyanosis, an increasing temperature (106.4° F.), and death after fifty-six hours. At autopsy brain was found to be normal; there was considerable bronchial secretion, but no pneumonia. Other organs were normal.[3]

CASE 6.—Child, aged three and a half, accustomed to small drinks of whisky. Said to have drunk at least 12 ounces (355 c.c.) of pure whisky in one afternoon. Fell into a kind of stupor lasting in varying degrees for more than two months. There were convulsions, right-sided paralysis, extreme contractures of the extremities due to multiple neuritis, strabismus, and repeated vomiting. Fever and symptoms of consolidation of the lower part of the right lung were also present. After about five months the child appeared to have recovered completely; there were no evidences that his mental condition had been impaired.[4]

Isolation.—Alcohol may serve as a type of a large group of substances whose common property of volatility with steam renders their separation from animal tissues a comparatively easy task. While the method employed is fundamentally a simple process of distillation, many complicated forms of apparatus have been devised, especially for the purpose of securing an effective fractional distillation, but also to avoid overheating the material or the loss of extremely volatile substances, such as chloroform and ether. Moreover, in the general application of the method it is necessary to observe certain precautions in order to prevent the occurrence of chemical alterations which might give rise to a confusion of the poison with its alteration products, or of deep-seated chemical decompositions in consequence of which the poison may altogether escape detection.[5]

But where the search is especially directed to alcohol, no serious difficulty can arise. After proper comminution the suspected material, if not already acid, is faintly acidified with tartaric acid and submitted to distillation by passing steam from a separate vessel. A simple and convenient form of apparatus is that represented in Fig. 24, p. 321.

The distillate is placed in an ordinary distilling flask, treated with pure magnesium oxid, and redistilled very slowly over the water-bath. This distillate is accurately measured, its specific gravity taken with a picnometer, and after a convenient portion has been removed for a quantitative determination, is used for the tests given below.

A large proportion of the water may be removed from the product without loss of alcohol by distilling over potassium carbonate. If a

[1] Kerr, *Lancet*, 1895, vol. i., p. 404.
[2] Devine, *Boston Med. and Surg. Jour.*, 1895, vol. cxxxiii., p. 545.
[3] Weber, *Edinburgh Med. Jour.*, 1897, vol. i., p. 631.
[4] Herter, *New York Med. Jour.*, 1896, vol. lxiv., p. 608.
[5] Consult also p. 321 in section on General Principles of Toxicology.

complete dehydration be desired,—a rather useless operation and one which necessarily involves serious loss of material,—it should be noted that the use of calcium chlorid is entirely inadmissible; that calcium oxid acts violently on dilute alcohol, and that the last traces of water can best be removed by digestion with dehydrated copper sulphate.[1]

FIG. 61.—Crystals of Iodoform.

Tests.—1. When treated with an aqueous solution of iodin in potassium iodid and then with enough 10 per cent. sodium carbonate solution to remove the brown color, alcohol yields, especially on warming, a yellow crystalline precipitate of iodoform[2] which is easily identified by its odor and by the various hexagonal forms which it exhibits under the microscope (see Fig. 61). This reaction is extremely sensitive and is not produced by ether nor by methyl-alcohol.[3] On the other hand, acetone, lactic acid, aldehyd, and a large number of substances will yield iodoform under the conditions stated.

2. On shaking an aqueous solution of alcohol with a drop of benzoyl chlorid and decomposing the excess of the reagent by warming with a little caustic soda, the odor of ethyl benzoate can be detected[4]—

$$C_2H_5OH + C_6H_5COCl + NaOH = C_6H_5CO_2C_2H_5 + NaCl + H_2O.$$

3. When warmed with dilute sulphuric acid and potassium bichromate, aqueous solutions of alcohol give off aldehyd,[5] which can be recognized by its odor, and the solution turns green from the formation of chromium sulphate—

$$3CH_3.CH_2OH + K_2Cr_2O_7 + 4H_2SO_4 = 3CH_3.CHO + K_2SO_4 + Cr_2(SO_4)_3 + 7H_2O.$$

If the escape of the aldehyd be prevented by working with a closed vessel, the alcohol is quantitatively oxidized to acetic acid, a reaction which forms the basis of an admirable quantitative method:[6]

$$3CH_3.CH_2OH + 2K_2Cr_2O_7 + 8H_2SO_4 = 3CH_3.COOH + 2K_2SO_4 + 2Cr_2(SO_4)_3 + 11H_2O.$$

4. Mix a little of the suspected solution with dry caustic potash and a few drops of carbon disulphid. When the excess of the latter has evaporated, add a drop of a neutral aqueous solution of ammonium molybdate and acidify with sulphuric acid. In the presence of alcohol a wine-red color will be produced.

[1] Soubeiran, *Liebig's Annalen*, vol. xxx., p. 356; Erlenmeyer, *ibid.*, vol. clx., p. 249.
[2] Lieben, *ibid.*, supp. 7, p. 218; Hager, *Zeitschr. f. anal. Chem.*, vol. ix., p. 492.
[3] Lieben, *Liebig's Annalen*, Supp. 7, p. 377.
[4] Baumann, *Ber. d. chem. Ges.*, vol. xix., p. 3218.
[5] Liebig, *Liebig's Annalen*, vol. xiv., p. 133.
[6] Bourcart, *Zeitschr. f. anal. Chem.*, vol. xxix., p. 609.

Since Bechamp[1] has shown that alcohol is to be found normally in traces in the animal body, a quantitative estimation should form a part of every toxicologic examination. For this purpose the method of Bourcart is preferable to the older ones which are based on the oxidation of alcohol to acetic acid with platinum black.[2]

An aqueous solution containing about 0.5 per cent. of alcohol is heated in a closed tube on the water-bath with sulphuric acid and a known quantity of a standard solution of potassium bichromate. The product is then treated with potassium iodid, and the liberated iodin titrated with a standard solution of sodium thiosulphate. By an obvious calculation the amount of reduced bichromate, and in turn the amount of alcohol, can be found.

FUSEL OIL.

A few cases of poisoning by fusel oil (impure amyl-alcohol, $C_5H_{11}OH$) have been reported; 2 such cases, with a review of the literature on this subject, are described by Futcher.[3] The chief symptoms were profound unconsciousness of several hours' duration, and glycosuria lasting two or three days. Glycuronic acid was also found in the urine. In one case methemoglobin was found in the urine.

FORMALDEHYD.

Formaldehyd (HCOH) is a colorless gas which has been used extensively in recent years as a germicide and as a hardening agent in anatomic work. Few cases of poisoning by formaldehyd have been reported as yet, but the number of such cases will probably be greatly increased in the near future as the public becomes better acquainted with the substance. Formaldehyd may be used either in the form of a vapor or as a solution in water; the latter is called formalin or formol, and the solution ordinarily used contains (or is claimed to contain) 40 per cent. of the gas.

The vapor of formaldehyd is very irritating to the mucous membranes, and workmen engaged in manufacturing it and those who use it in disinfecting houses often suffer severely from coryza, conjunctivitis, and bronchitis. Moreover, it has been found that animals are killed when confined in a room the air of which contains the vapor in sufficient quantity to act as an efficient germicide.[4]

Formalin is used very extensively as a preservative for milk and other articles of food; there has been much discussion as to the effect of the quantities ordinarily used for this purpose upon the individuals taking such articles of food.[5] It has been shown conclusively that small quantities of formaldehyd greatly delay the digestion of proteids by gastric and pancreatic juice, the digestion of starch by the pancreatic juice, and the curdling of milk by rennet.[6] It is also known that some

[1] Bechamp, *Zeitschr. f. anal. Chem.*, vol. xx., p. 603.
[2] See *Foods*, A. W. Blyth. [3] *Amer. Med.*, vol. ii., p. 210.
[4] Harrington, *Amer. Jour. Med. Sci.*, 1898, vol. cxv., p. 69.
[5] See *Lancet*, 1900, vol. i., pp. 207, 279, 429.
[6] Bliss and Novy, *Jour. of exp. Med.*, 1899, vol. iv., p. 47; Halliburton, *Brit. Med. Jour.*, 1900, vol. ii., p. 1.

individuals are especially susceptible to the effect of formalin, small quantities added to food causing dyspepsia and other disturbances of digestion. Annette[1] found that young animals suffered severely from the effects of small quantities of formalin; thus many kittens died when fed on milk containing 1 part of formaldehyd in 2500 or even 50,000 (the quantities ordinarily used to preserve milk), while others grew very slowly and suffered greatly from malnutrition. On the other hand, Tunnicliffe-Rosenheim[2] failed to find any injurious effects in children whose food contained 1 part of formaldehyd in 9000. There are few, if any, preservatives that are less injurious than formalin, and circumstances may arise when it would be better to use formalin as a preservative than to run the risk of using milk that is undergoing decomposition. Dealers should be compelled, however, to label distinctly milk so preserved, and such milk should not be given to young children. In some States the use of formalin as a food preservative is prohibited by law, and there is no doubt that it is far safer to preserve food by cold and attention to cleanliness than by the addition of any chemical.

A few cases of severe poisoning are reported in which formalin was taken internally. Thus Bock[3] reports a case in which an imbecile, aged twenty-six, swallowed from 1 to 3 ounces (30 to 89 c.c.) of a 4 per cent. solution of formaldehyd. The symptoms were pain and vomiting; the vomitus was blood-stained and had the odor of formaldehyd. Free emesis was produced by apomorphin. The vomiting continued for several hours. After sixteen hours some weakness of the pulse was noticed, but there was little pain or nausea. There were no mental symptoms or cyanosis, but the cardiac depression continued and led to death thirty-two hours after the poison was taken. At the autopsy, made one hour after death, no formalin was found in the stomach. The stomach-walls were edematous, the cardiac end red and highly inflamed; the remainder of the stomach-wall, including the muscular coat, was "necrotic, dark, tough, and cut like old leather." Some inflammation of the duodenum was noticed. In a case reported by Klüber, in which a man drank an unknown quantity of formalin in Apenta water, the chief symptoms were similar to those of acute alcoholic intoxication;[4] there was profound stupor, from which the patient could not be aroused. Later there was a condition of excitement, followed by headache. The eyelids became red, and there was profuse lacrimation. No urine was passed for nineteen hours; the first which was evacuated reduced a solution of silver nitrate. Recovery was complete. Similar symptoms, with recovery, followed the swallowing of 60 to 70 c.c. (2 to $2\frac{1}{3}$ ounces) of 35 per cent. solution of formaldehyd.[5]

Zorn[6] reported a case in which a man recovered after drinking about

[1] Annette, *Lancet*, 1899, vol. ii., p. 1282.
[2] Tunnicliffe-Rosenheim, *Jour. Hyg.*, vol. i., p. 311.
[3] Bock, *Indiana Med. Jour.*, 1899, vol. xviii., p. 122.
[4] Klüber, *Münch. med. Wochenschr.*, 1900, vol. xlvii., p. 1416.
[5] Gerlach, *ibid.*, 1902, p. 1503
[6] Zorn, *ibid.*, p. 1588. This case, also that of Klüber, is abstracted in the *Brit. Med. Jour.*, 1901, vol. i., Epitome No. 42.

half an ounce (15 c.c.) of what was probably the ordinary 40 per cent. solution of formaldehyd; the chief symptoms came from irritation of the alimentary tract and the kidneys. There were retching, vomiting, vertigo, dyspnea, and a burning pain in the mouth and stomach. There was diarrhea, with violent tenesmus. The secretion of urine was suppressed for twenty-four hours. Recovery was complete in four days.

In a case of poisoning by formalin the administration of a dilute solution of ammonia seemed to give good results.[1]

It is evident, from the above cases, that the symptoms in formaldehyd poisoning vary considerably.

Aqueous solutions containing 1 part of formaldehyd in 2000 are very irritating to the skin; long-continued application of stronger solutions leads to ulcerations and gangrene; weak solutions lead to eczema.[2]

Detection.—The material under examination is made into a paste with water that has been acidified with sulphuric acid and heated at 80° C. for a short time. An excess of dry powdered sodium sulphate is added, and the material is submitted to distillation. All the formic aldehyd will pass over in the early portions of the distillate and may be shown by the following tests:[3]

1. Upon the addition of a few drops of the distillate to a fuchsin solution that has been decolorized with sulphur dioxid the presence of the aldehyd is shown by the appearance of a red color. The test is given by aldehyds in general.

2. Acidify a portion of the distillate with sulphuric acid, add a few drops of dimethylanilin, and heat at 40° C. for an hour or two in a sealed tube. Make the product alkaline with sodium carbonate and evaporate the excess of dimethylanilin. Acidify with acetic acid, and add a trace of lead dioxid, when the presence of formic aldehyd will be shown by the production of an intense blue color.[4]

3. To a portion of the distillate are added twice its volume of alcohol and a few drops of diphenylenedihydrazin hydrochlorate. On warming, the solution is colored yellow, and on standing a while a yellow crystalline precipitate is formed.[5] This reaction is characteristic of formic aldehyd.

4. Heat a portion of the distillate with an equal volume of 40 per cent. caustic potash which contains 5 per cent. of resorcin. The presence of formic aldehyd is shown by the production of a red color.[6]

PARALDEHYD.

Acute poisoning with paraldehyd is very rare; at least one person, however, has died from an accidental overdose,[7] and another apparently used it as a means of committing suicide.[8] Cases of chronic poisoning

[1] André, *Jour. de Pharm. et Chimie*, 1899, 6 s., vol. x., p. 10.
[2] Fisher, *Brit. Jour. Derm.*, vol. xiii., p. 306.
[3] Jean, *Chem. Centralbl.*, 1899, vol. i., p. 641.
[4] Trillat, *ibid.*, 1898, vol. ii., p. 585
[5] Neuberg, *Ber. d. d. chem. Ges.*, vol. xxxii., p. 1962.
[6] Lebbin, *Chem. Centralbl.*, 1899, vol. i., p. 641.
[7] *Lancet*, 1890, vol. ii., p. 423. [8] Paltauf, *Wien. klin. Wochenschr.*, 1893, p. 888.

with paraldehyd are more common, as some individuals use it as others do morphin, chloral, etc.

Properties.—Paraldehyd (*Paraldehydum*, U. S. P., $C_6H_{12}O_3$), a polymeric form of ethyl aldehyd, is a colorless liquid that has a strong and characteristic odor and a burning, disagreeable taste. It is soluble in water, alcohol, and ether, solidifies when subjected to cold, the crystals thus formed melting at 10.5° C. (51° F.).

The physiologic action of paraldehyd is very similar to that of chloral, but it causes less depression of the circulation and respiration. The drug is eliminated by the lungs and kidneys; it imparts a very characteristic and disagreeable odor to the breath. Tolerance for paraldehyd is rapidly established.

Symptoms.—After an average dose the effects are those of the simple hypnotics, with little cardiac or respiratory depression. Larger doses occasionally cause nausea, vomiting, headache, and dizziness. After a very large dose there are deep stupor and complete relaxation of the muscles, as seen in persons under the influence of chloroform; the face is flushed, and the pupils are somewhat contracted. This condition may continue for many hours (thirty-four in one case[1]) and recovery take place.

The symptoms in chronic paraldehyd poisoning are various. There are usually disturbances of the digestion, emaciation, general muscular weakness, and mental failure, with tremors of the hands and tongue. Skin eruptions similar to those caused by chloral are common. There is usually insomnia, and there may be discontent, confusion, temporary loss of memory, hallucinations, and delusions of persecution.[2] The symptoms resemble in some respects those of chronic alcoholism, and a condition not very unlike that of delirium tremens results.

Little is known as to the **fatal dose** and the **fatal period** of paraldehyd. Six or 7 drams (22.5 to 26 c.c.) given to a patient delirious from typhoid fever caused unconsciousness in five minutes and death in four hours.[3] Three and a half ounces (104 c.c.) caused severe, but not fatal, poisoning.[4] Those accustomed to the use of paraldehyd can take enormous doses: thus a woman learned to take from 16 to 18 ounces (473 to 532 c.c.) of the "elixir of paraldehyd" in twenty-four hours.[5] Doses of 1 ounce (30 c.c.) have been taken for months without causing any symptoms; on the other hand, 2 ounces (59.2 c.c.) a day have led to the most severe symptoms.[6]

The **treatment** of both acute and chronic poisoning by paraldehyd is on the same general lines as that of poisoning by chloral.

The **postmortem appearance** of the stomach seems to resemble that caused by rather dilute alcohol;[7] the odor of the poison or of its decomposition-products may be noticed.

[1] MacKenzie, *Brit. Med. Jour.*, 1891, vol. ii., p. 1254.
[2] Elkins, *Quarterly Journal of Inebriety*, 1894, p. 333.
[3] *Lancet*, 1890, p. 423.
[4] MacKenzie, *loc. cit.*
[5] Goodman, *American Practitioner and News*, vol. x., p. 289.
[6] Reinhold, *Therap. Monatsh.*, 1897, p. 300.
[7] Paltauf, *loc. cit.*

Detection.—The odor of paraldehyd or of its decomposition-products may be noticed. The material under examination is acidified with sulphuric acid and subjected to distillation with steam. Under these conditions paraldehyd is converted into ordinary aldehyd,[1] and the receiver should, therefore, be surrounded with ice. Aldehyd in dilute solution, as it will be obtained in this manner, may be shown by the following tests:

1. A fuchsin solution that has been decolorized with sulphur dioxid becomes violet red when treated with a dilute solution of aldehyd in the cold.[2] This reaction is general for the aldehyds, but is produced also, though slowly, by some ketones.

2. On treatment with ammoniacal silver nitrate in the cold, dilute solutions of aldehyd cause the deposition of a mirror of silver:[3]

$$2AgONH_4 + CH_3CHO = CH_3.COOH + 2NH_3 + H_2O + 2Ag.$$

With Tollens' reagent[4] the presence of 1 part of aldehyd in 1000 parts of water can easily be shown.

CHLORAL HYDRATE.[5]

Most cases of poisoning with chloral are accidental. Of 93 cases collated by Witthaus,[6] 70 were accidental, 22 suicidal, and 1 homicidal. Of 127 deaths due to chloral in England and Wales from 1883 to 1892, 111 were accidental, 15 suicidal, and 1 due to murder. The earlier accidental cases were due, for the most part, to physicians prescribing the drug in too large doses or to their administering it to patients having weak hearts; the later ones to patients taking the drug without professional advice, or to mistakes. Chronic chloral poisoning is not uncommon; it usually results from too long-continued use of medicinal doses. Chloral has been used as "knock-out drops" for the purpose of effecting robbery[7] or rape. In most cases of poisoning the drug has been taken by the mouth, but death has resulted from its introduction into the rectum and from its injection into a vein for the purpose of producing surgical anesthesia.

Properties.—Pure chloral ($CCl_3.CHO$) is an oily liquid and is seldom seen. Chloral hydrate (*Chloral*, U. S. P., *chloral hydras*, $CCl_3.CHO.H_2O$) is formed by mixing chloral with water; it occurs in the form of granular, sugar-like crystals. It is slightly volatile at ordinary temperatures, melts at about 58° C. (136.4° F.), and boils at 97.5° C. (207.5° F.). It is easily soluble in water, alcohol, and ether. It has a peculiar odor, often compared to that of a melon, and a dis-

[1] Weidenbusch, *Liebig's Annalen*, vol. lxvi., p. 152.

[2] Caro, *Ber. d. d. chem. Ges.*, vol. xiii., p. 2342 (foot-note); Schmidt, *ibid.*, vol. xiv., 1848.

[3] Tollens, *ibid.*, vol. xiv., p. 1950.

[4] To a solution of 3 gm. of silver nitrate in 30 gm. of 25 per cent. ammonia add 3 gm. of caustic soda in 3 c.c. of water (Tollens, *ibid.*, vol. xv., p. 1635).

[5] This substance is usually termed chloral, and is so designated in the U. S. P.; by "chloral" in this article is meant chloral hydrate, unless the contrary is stated.

[6] Witthaus and Becker, *Medical Jurisprudence, Forensic Medicine, and Toxicology*, vol. iv., p. 855.

[7] Case 4.

agreeable taste; the latter is almost absent when the drug is dissolved in alcoholic beverages. Chloral is rapidly decomposed by alkalis into chloroform, formates, and water, at ordinary temperatures.

Physiologic Action.—The action of chloral is essentially the same as that of alcohol and chloroform—*i. e.*, it depresses and eventually paralyzes the central nervous system. There is seldom, however, a period of excitement with chloral, and the sensation of pain is not greatly dulled until very large doses have been administered. The chief effect of small doses is to cause sleep that closely resembles natural sleep. With larger doses the sleep is more profound and the patient can no longer be aroused to complete consciousness; the reflexes are lessened, the respiration and heart are slowed, and the blood-pressure is lowered. With very large doses the effect is almost exactly similar to that observed in the third stage of anesthesia from chloroform; reflexes and sensation are completely abolished and the muscles are relaxed. The prolonged administration of chloral leads to fatty degeneration of various organs. Locally, chloral is an irritant; it also possesses antiseptic properties. Chloral is excreted in the urine partly as chloral, but chiefly in combination with glycuronic acid; this compound reduces Fehling's solution, and so may be mistaken for sugar. Chloral is not decomposed by the blood into chloroform to any considerable extent.

Symptoms.—The symptoms of chloral poisoning vary widely in different cases. In some cases, especially in conditions of weakened heart action, as in delirium tremens, the insomnia of continued fevers, etc., death may result very rapidly from failure of the heart;[1] in some cases it occurs even before drowsiness is noticed. In a few cases there is a period of excitement[2] and the individual may act like an intoxicated person. There may be nausea and vomiting, due to the local action of the drug upon the stomach. In most cases the patient falls into a deep sleep from which it is impossible to arouse him; the pulse is scarcely perceptible; the respiration is irregular and shallow; the pupils are moderately contracted, rarely dilated; the face is cyanotic or, in the earlier stages, flushed, on account of the dilatation of the vessels; the extremities are cold. There is usually a fall of temperature from the beginning: it may sink to 86° F. In rare cases a rise of temperature has been noted.[3] Death usually occurs in coma and seems to be due, as a rule, to failure of the respiration; sometimes it is due to pulmonary edema. Occasionally convulsions and delirium occur. The patient may apparently recover, but die later, or various symptoms may continue for some time.[4]

The symptoms of chronic chloral poisoning are various, and the con-

[1] Case 1. [2] Case 2.

[3] Plummer, *Lancet*, 1894, vol. i., p. 21; also Case 3. Possibly the large doses of strychnin administered in some of these cases contributed to the elevation of the temperature, but usually some rise was noted before the strychnin was injected.

[4] Rogers, *Medical Record*, vol. lvii., p. 412 (recovery from 680 grains—44 gm.—taken in three days); see also Case 3.

dition is often difficult to diagnose. There are frequently disturbances of digestion, with diarrhea and loss of weight. Affections of the skin are common; these may take the form of a scarlatina-like rash, of vesicles, or of a superficial ulceration at the roots of the nails. There is frequently much irritation of the eyes, as shown by itching, redness, swelling, and excessive secretion. These symptoms may follow a single large dose or a number of small ones. The nervous symptoms are marked: sleeplessness, indefinite pains, general depression with impairment of the mental faculties are often observed. In some cases a condition resembling melancholia results, while in others it is difficult to distinguish the condition from paretic dementia.[1] Sudden withdrawal of the drug sometimes leads to symptoms resembling the delirium tremens caused by alcohol. Sudden death is common among habitual chloral takers; it seems to be due to failure of the heart, and may occur after doses but slightly larger than those to which the patient is accustomed.

Fatal Dose.—The fatal dose of chloral seems to be dependent to a considerable degree upon the age and condition of the individual. Children have been said to bear chloral better, in proportion, than do adults, but 3 grains (0.194 gm.) have been fatal to an infant a year old and 46 grains (2.98 gm.) to a child of five years. Although much larger doses are frequently given and may be necessary in cases of tetanus and in some other conditions, 30 (1.94 gm.) and even 20 grains (1.29 gm.)[2] have on numerous occasions caused the death of adults. A patient suffering from epileptic mania recovered from 1.1 ounces (34.2 gm.), and in a case of tetanus 3 ounces (93.3 gm.) were given in twenty-four hours without causing death.[3] A woman in good health and with appropriate treatment ultimately recovered from 1 ounce (31 gm.) of chloral.[4] Recovery has frequently followed the taking of from 160 to 180 grains (10.36 to 11.66 gm.); after such large doses the recovery has doubtlessly been due in many cases to the prompt treatment. One hundred and twenty grains (7.77 gm.) would probably be very dangerous to most adults in good health, and 150 grains (9.71 gm.) fatal;[5] much smaller quantities would be fatal to most aged individuals and to those having a weak heart. The late Professor Tyndall died from the effects of taking 80 grains (5.18 gm.). H. C. Wood thinks 20 grains (1.29 gm.) is the highest safe dose of chloral, and that this should not be repeated more than twice, at intervals of, at least, an hour, except in very unusual cases.

Fatal Period.—The fatal period is variable, and seems to be partly dependent upon the size of the dose. In one fatal case 30 grains (1.94 gm.) caused death in thirty-five hours;[6] in others, doses of 75 grains (4.85 gm.) have caused death in from fifteen minutes to one hour. Perhaps in most of the recorded cases death has occurred in from six to ten hours. Yet death has taken place within ten minutes

[1] Case 5.
[2] *Practitioner*, 1877, vol. xix., p. 116.
[3] Case 1.
[4] Case 3.
[5] Blyth, *Poisons*, p. 159.
[6] Case 2.

after taking from 20 to 30 grains (1.29 to 1.94 gm.). In several of the very rapidly fatal cases there has been disease or weakness of the heart. In a number of cases death has occurred suddenly after the last of several small doses.

Treatment.—The stomach should be washed out with warm water; emetics, with the exception, perhaps, of apomorphin, often fail to cause vomiting on account of the great depression of the nervous system. If, for any reason, it is not possible to wash out the stomach, the administration of an alkali may be of use; some of the chloral may be destroyed in this manner.[1] Chloral is absorbed so rapidly that in many cases probably little or none of the drug is in the stomach when the patient is seen by the physician; hence physiologic antidotes are necessary. Of these, strychnin hypodermically and drafts or rectal injections of hot coffee seem to give the best results. Atropin is also recommended. Artificial respiration is sometimes necessary, and the inhalation of oxygen seems to be of value. The temperature of the patient should be maintained by the use of blankets, hot-water bottles, etc.

In chronic chloral poisoning the drug should be rapidly withdrawn, and stimulants, such as strychnin and digitalis, given. Special attention should be paid to the diet. In many cases the treatment is successful only in special hospitals.

Postmortem Appearances.—No characteristic lesions are caused by chloral. The brain is said to be, as a rule, hyperemic, and the blood to be dark and fluid. The mucous membrane of the throat, esophagus, and stomach may be reddened and inflamed. The contents of the stomach may have the odor of chloral. Occasionally there is edema of the lungs. In chronic cases of chloral poisoning fatty degeneration of the heart is common; fatty degeneration or fatty infiltration of the kidneys has been produced experimentally in animals by the administration of chloral.

Cases of Poisoning by Chloral Hydrate.

Case 1.—Woman, aged thirty-three, wished to have teeth extracted and was given 10 grains (0.65 gm.) of chloral to prevent pain. A second dose of 10 grains (0.65 gm.) was given an hour later; in a few minutes symptoms of poisoning appeared and death occurred in about fifteen minutes.[2]

Case 2.—Woman, aged twenty, given at 10 P. M. 30 grains (1.95 gm.) of chloral for sleeplessness. Caused pain and burning in chest and some excitement. In the morning was found very pale and unconscious; could not be aroused. Labored respiration; no pulse at wrist; extremities were cold; pupils dilated. This condition continued practically unchanged, notwithstanding that various stimulants were administered, until death occurred about thirty-five hours after the dose was given. There was at no time any return of consciousness or any movements.[3]

Case 3.—Woman in excellent health, aged thirty-four, took at 8 A. M. 1 ounce (31 gm.) of chloral dissolved in 2 ounces (59.2 c.c.) of water. At 4 P. M. she was discovered in bed unconscious, and it was impossible to arouse her. When seen by physician at 5.30 P. M. she was comatose and all reflexes were abolished. Respiration shallow and stertorous; pupils small and sluggish; pulse 130; tempera-

[1] Dougall, *Glasgow Med. Jour.*, 1895, p. 95.
[2] Ingals, *Chicago Med. Jour. and Exam.*, 1877, vol. xxxiv., p. 234.
[3] Fuller, *Lancet*, 1871, vol. i., p. 403.

ture 100.5° F. Atropin and later strychnin and ether were given hypodermically. The stomach was washed out with difficulty. Nitrite of amyl by inhalation improved the pulse. Enemata of strong coffee were given at 10 P. M.; sinapisms were applied to legs, and patient was vigorously rubbed with towels. At midnight the temperature was 103° F., pulse was flagging, and the prognosis was grave. Enemata of coffee, milk, and brandy and hypodermic injections of strychnin were continued. At 12.30 A. M. she showed the first signs of animation—twitchings of face, movements of limbs, and soon groaning and restlessness. Imperfect consciousness soon returned, but patient slept most of the day. Nine-tenths of a grain (0.06 gm.) of strychnin had been given. The temperature did not fall for a day or two. Patient did not leave room for over three weeks, and for some time afterward there was impairment of digestion and muscular tone, and periods of excitement alternated with periods of great depression for six weeks.[1]

CASE 4 (The "Manchester Cab Mystery.").—J. F., a man weighing about 15 stones (210 lbs.), had been drinking during the day and by evening was partially intoxicated. At 6.20 he drove to a public house with a young man and had a glass of beer. Reëntered the cab at 7.10. He is said to have walked quite steadily and seemed to be sober. At about 7.25 cabman found the cab-door open. J. F. was alone, his head had fallen forward on to the front seat, he was unconscious but could be roused, and could just speak. All his valuables were gone. He died at 8.05, death being due immediately to syncope. Autopsy nineteen hours after death. Organs had a strong odor of alcohol. Heart showed slight fatty degeneration. Stomach slightly congested. Liver large, fatty, and cirrhotic. Traces of chloral were found in the contents of the stomach and upper part of the small intestine. It was proved that the young man who had accompanied J. F. had twice used chloral for drugging persons, and that he had put something into one of the glasses of beer at the public house. He was convicted of murder in the first degree.[2]

CASE 5.—Lady, aged fifty-six, had been ill for five years. For a year and a half had been taking a proprietary medicine containing chloral; recently the dose had been increased, but the total amount did not exceed 20 grains (1.29 gm.) of chloral a day. She had delusions, transitory in character and of a grandiose form. Ataxia, especially of the lower limbs, was marked. General tremor was present, pupils were unequal, and speech was tremulous. There was marked insomnia. Had been violent at times and had delusions of persecution. Diagnosis of paretic dementia was made, and patient was about to be sent to an insane asylum. All hypnotics, however, were withdrawn, and in a few days her delusions began to wane and her nervous state to improve. Tremors lessened, speech sharpened, and the pupils reacted equally. Six weeks after the withdrawal of the chloral she went home and resumed the management of her estate.[3]

H. H. Kane[4] gives a summary of 63 cases of poisoning by chloral; the symptoms, fatal dose, etc., are fully discussed.

Isolation.—In dealing with organic matter supposed to contain chloral one must bear constantly in mind that this substance is decomposed by caustic alkalis, giving rise to chloroform and a formate; but as very dilute alkalis are inefficient in effecting this decomposition, chloral may be recovered from a fluid that is initially faintly alkaline.[5] In such a case the material should be acidified with tartaric acid as soon as possible.

The method of distillation with steam described under alcohol is peculiarly useful for the isolation of chloral, since by the ordinary

[1] Colenso, *Lancet*, 1894, vol. ii., p. 1034.
[2] Reynolds, *Brit. Med. Jour.*, 1889, vol. ii., p. 235.
[3] Coe, *Quarterly Journal of Inebriety*, vol. xxi., p. 65.
[4] *Medical Record*, 1880 and 1881, vols. xviii., xix.
[5] Chloral hydrate is decomposed instantly by normal caustic soda in the cold, but by tenth normal alkali only slowly in the warm. Meyer and Haffter (*Ber. d. d. chem. Ges.*, vol. lxi., p. 600).

method it is necessary to evaporate, almost to dryness, in order to obtain the greater portion of the poison. To the dilute aqueous distillate the following tests may be applied.[1]

Tests.—1. The liquid will respond to many of the tests for chloroform, but possesses no odor of chloroform.

2. When shaken with ether in a separating funnel, chloral passes into this solvent and can be recovered as a solid residue upon evaporation. This residue is not inflammable, whereas chloral alcoholate ($CCl_3.CHO.C_2H_5OH$) burns readily.

3. On the addition of a few drops of 20 per cent. sodium hydroxid to a portion of the fluid obtained in the earlier stages of the distillation, the liquid becomes cloudy and drops of chloroform soon collect on the bottom of the vessel. This substance may be identified by the tests given under chloroform.

4. To a rather large amount of the distillate is added some magnesium oxid, and the fluid is boiled in a flask provided with a long condensing tube:

$$2CCl_3.CHO.H_2O + MgO = 2CHCl_3 + (HCO_2)_2Mg + H_2O.$$

After heating on the water-bath to expel the chloroform, the liquid is made acid with tartaric acid, and the formic acid is distilled off and identified by appropriate tests.

(*a*) On boiling a solution of formic acid with acidified potassium permanganate, the latter is reduced and the color of the solution disappears.

(*b*) When heated with a solution of mercuric chlorid, formic acid causes the formation of insoluble mercurous chlorid:

$$2HgCl_2 + H.CO_2H = 2HgCl + 2HCl + CO_2.$$

Trichloracetic acid also yields chloroform when treated with alkalis, but no formic acid is produced:[2]

$$CCl_3.COOH + 2NaOH = CHCl_3 + Na_2CO_3 + H_2O.$$

Estimation.—The amount of chloral hydrate present in an aqueous solution can easily be determined.[3] The liquid is shaken with magnesium oxid to remove any free acid, and treated with an excess of standard sodium hydroxid (must be as strong as a normal solution). After the excess of alkali has been determined with a standard acid, the amount of chloral hydrate can be found by an obvious calculation—1 c.c. of normal alkali corresponds to 0.1655 gram of chloral hydrate.

CHLOROFORM.

Most of the deaths from chloroform result from its use as an anesthetic in surgical and medical practice; it is estimated that on an average one death occurs in about 3000 inhalations, but the figures in

[1] The various methods of extraction which have been recommended for chloral involve great loss of material and are inferior in every way to the one described.

[2] Beckurts and Otto, *Ber. d. d. chem. Ges.*, vol. xiv., p. 589; Seubert, *ibid.*, vol. xviii., p. 3342.

[3] Meyer and Haffter, *loc. cit.*

different statistics vary widely. Chloroform is occasionally used as a means of committing suicide; in these cases the liquid is frequently, probably usually, taken internally. Occasionally chloroform or chloroform liniment is taken internally by mistake or an overdose is taken. It is sometimes used as an intoxicant, being either taken internally or inhaled. Chloroform is rarely used for the purpose of murder. By proceeding very cautiously it is possible to chloroform a person while asleep,[1] but, as a rule, the person awakes before anesthesia begins. There seem to be no authentic cases of the use of chloroform in this manner for criminal purposes.

Properties.—Chloroform (*Chloroformum*, U. S. P., trichlormethane, $CHCl_3$) is a heavy, colorless liquid, of a characteristic ethereal odor, a burning, sweet taste, and a neutral reaction. Soluble in something over 200 parts of water; mixes in all proportions with ether and with five volumes or more of alcohol. Specific gravity, 1.491; boiling-point, 62° C. (143.6° F.). Alcohol, ethyl chlorid, aldehyd, chlorin, hydrochloric acid, and other impurities are sometimes present; the toxicity of chloroform is somewhat enhanced by these. Chloroform and its vapors are not inflammable, but when it is used for a long time, as in obstetric or surgical cases, near a flame, decomposition-products are formed that are very irritant; in fact, fatal poisoning has resulted from these products.[2]

Physiologic Action.—Locally, chloroform is an irritant; hence its use as a liniment. Taken by the mouth it causes pain, vomiting, and frequently diarrhea. When inhaled,—the usual method of administration,—the local action of the vapor upon the mucous membranes of the air-passages is often very marked and consists of a reflex slowing or stoppage of the respiration, a reflex rise of blood-pressure, and a slowing of the heart; these reflex actions are usually of but brief duration.

When chloroform reaches the circulation, the effects are similar to those of alcohol. There is a period of excitement, due either to a stimulation of certain parts of the central nervous system or more probably to a paralysis of certain controlling centers. After the period of real or apparent stimulation comes a stage of depression; the higher centers of the brain are first affected, then the spinal cord, and last of all the medulla. The sensory functions of the brain and cord are depressed earlier than are the motor. Respiration becomes slow from the depression of the respiratory center; the heart beats more weakly from a direct action of the poison on the cardiac muscle, and the blood pressure sinks. Chloroform has a special affinity for the red blood-corpuscles; a large amount is also absorbed by the brain. The temperature falls during even a short period of anesthesia. Fatty degeneration of the liver, heart, and kidneys follows the repeated administra-

[1] Kelly, *Medical Record*, 1890, vol. i., p. 352. See also *Therap. Gaz.*, 1894, p. 758; *Medical Record*, 1890, vol. i., pp. 42, 139.

[2] *Ibid.*, 1899, p. 601. See Gerlinger, *Arch. f. exper. Path. u. Pharm.*, 1902, vol. xlvii., p. 438.

tions of chloroform or sometimes a single prolonged administration.[1] Chloroform is largely eliminated by the lungs; some seems to be destroyed in the body, the chlorin appearing in the urine as chlorids.

Symptoms.—It is customary to recognize three stages in the symptoms produced by chloroform. The first stage is that of excitement. There is a feeling of warmth,—first of the face, then of the entire body,—followed by a tingling sensation of the skin. There are ringing and roaring sounds in the ears, and vision becomes distorted. Consciousness is soon lost, and the patient may struggle violently and indulge in profane or abusive language. The second stage is that of surgical anesthesia: the muscles are relaxed, the patient lies perfectly still, with regular, but rather slow and shallow respiration, and is entirely insensible to pain. This stage may be maintained for hours. If the inhalation of chloroform be discontinued at this stage, the patient usually awakes within from twenty to forty minutes, but he may not awake for some hours. Vomiting, which may occur at almost any stage, may persist for some time. There is usually some confusion of ideas also. The third stage is that of paralysis, and is characterized by the failure of the respiration and heart. The skin becomes dark and cyanotic, and the pupils dilate widely. There has been a discussion in progress for many years as to the cause of death in chloroform inhalation; some maintain that it is due to failure of the heart, others to failure of the respiration. It seems probable that in most cases in which death occurs late in the anesthesia the respiration ceases first; in many cases life may be maintained for some time by means of artificial respiration, the heart continuing to beat fairly well. In other cases the heart is so deeply poisoned that it is unable to beat long after the respiration has ceased, although artificial respiration is maintained; this occurs most frequently when the chloroform vapor is inhaled in concentrated form. Many of the deaths which occur early in the administration of chloroform are attributed by Embley[2] to stoppage of the heart by the direct action of the poison upon the cardio-inhibitory center.

Probably the most frequent cause of death in chloroform anesthesia is the inhalation of the vapor in too concentrated a form; a large quantity of the chloroform reaches the blood suddenly and causes immediate paralysis of the respiration and heart or stoppage of the latter through stimulation of the vagus. A lethal quantity of chloroform may be absorbed from a few deep inhalations. The condition of the patient is also frequently a factor in the fatal result. It is dangerous to administer chloroform to the very old, to those suffering from fatty heart, atheromatous arteries, diabetes, and chronic diseases of the kidneys. Many fatalities occur among drunkards: it has been stated that the mortality among these has been as high as from 10 to 13 per cent. Death occasionally results from suffocation due to the drawing into the air-passages of vomited matter. Some deaths, especially

[1] Frankel, *Virchow's Archiv*, 1892, vol. cxxvii., p. 381; Strassmann, *Berlin. Klinik*, Feb. 1, 1898.

[2] Embley, *Brit. Med. Jour.*, April 5, 12, and 19, 1902.

in children, seem to be due to the chloroform containing impurities. Fatty degeneration of the heart and kidneys sometimes leads to death days after the chloroform was administered.

Death occasionally occurs under chloroform when every precaution is taken; as such cases occurred in operations before the introduction of anesthetics, it seems very probable that frequently death is not due to the anesthetic at all.[1]

The symptoms following the taking of chloroform internally are due partly to the local irritant action, partly to the effects after absorption. The local irritant action of the poison leads to intense pain in the throat and abdomen, and the patient may throw himself about violently. Vomiting usually results, and there is sometimes involuntary evacuation of the bowels. The chloroform is rapidly absorbed, and the subsequent symptoms may resemble those caused by the inhalation of the drug. With small doses there may be a period of excitement and the patients stagger like a drunkard; with large doses the depression begins at once. In most of the recorded cases unconsciousness seems to have come on within ten minutes. The skin becomes cyanotic and cool, the respiration irregular and stertorous, and the breath smells of chloroform; the pulse is small and weak; the pupils may be dilated or contracted, or pass rapidly from the one state to the other; vomiting soon ceases, owing to the depression of the nerve-centers; with most "irritant" poisons it is very persistent. Death may occur in coma, but it seems due in many cases to gastritis. Coma may continue for many hours and recovery still take place, but abdominal pain, bloody diarrhea, swelling of the liver, jaundice, and painful micturition may continue for several days. Death may occur after some time from pulmonary edema, failure of the heart, or, more frequently, from gastritis. The death-rate in cases in which chloroform has been swallowed is high. In a series of 57 cases collected by Eliot,[2] 15 died, while of 17 cases reported by Hirsch,[3] 7 died. Thus the death-rate has been from 26 to 41 per cent.

There is also a chronic form of chloroform poisoning due to the individual inhaling or drinking the drug; this habit is frequently the result of the use of small amounts for the relief of pain, and is sometimes associated with other drug habits. The chief symptoms are due to disturbances of the digestive and nervous systems. The patient may have hallucinations or pass into a condition that cannot be distinguished from the delirium tremens of alcoholism.

Fatal Dose.—It is impossible to make any definite statements as to the fatal dose of chloroform when taken by inhalation: it is far more a question of the concentration of the vapor than of the absolute amount inhaled. Air containing 1.5 per cent. of chloroform causes death in animals in one to two hours, and the rule is frequently laid down that patients should never, even for a short time, receive air containing more than 3.5 to 4 per cent. of chloroform vapor. Cases have been reported in which 30, and even 15, drops of chloroform have caused death: on the other

[1] See Case 2. [2] Eliot, *Medical Record*, 1885, vol. xxvii., p. 29.
[3] Hirsch, *Zeitschr. f. klin. Med.*, 1894, vol. xxiv., p. 190.

hand, 22½ ounces (997 gm.) have been inhaled in twenty-four and even in twelve hours, and the patient recovered. The fatal dose of chloroform when taken per os is also difficult to determine. A child of four years died from 1 dram (5.6 gm.), while another of the same age recovered from nearly twice as much. An adult died from about 4 drams (22.5 gm.), while another recovered from 5 ounces (222 gm.).[1] Forty minims (3.7 gm.) have caused severe, but not fatal, poisoning.[2] In most of the recorded fatal cases from ½ to 2 ounces (22.5 to 88.5 gm.) of chloroform have been taken; as a rule, death will almost certainly follow the administration of 11 or 12 drams (61.5 or 67.5 gm.).

Fatal Period.—Death may occur at any stage of chloroform inhalation. In about one-half of the reported cases death has occurred before anesthesia was fully produced—*i. e.*, within ten or fifteen minutes;[3] occasionally consciousness returns and death takes place a day or two later. When chloroform is taken internally, death may occur within an hour or it may be delayed for several days; in two cases it occurred eight days after the chloroform was taken. In most of the recorded cases death has occurred after the first twenty-four hours.

Treatment.—In cases of the failure of the respiration during the inhalation of chloroform the administration of the anesthetic should be immediately stopped. The head should be lowered and the feet raised so as to drive the blood to the brain. Artificial respiration should be commenced. If the heart is weak, rhythmic pressure over the heart may be practised. Many drugs have been recommended, but it is doubtful if any of them are of much use at this stage; perhaps the intravenous administration of the active principle of the suprarenal gland (epinephrin or "adrenalin") will prove of value. The administration of oxygen is also recommended. The treatment when the chloroform has been taken internally is that of an irritant and narcotic poison. The stomach should be washed out with warm water; emetics may be used, but they are somewhat uncertain in their action in these cases. Much relief is obtained from the use of demulcent drinks, such as olive oil. Strychnin, digitalis, atropin, ammonia, hot coffee, etc., may be used as stimulants; cold affusions and, in extreme cases, artificial respiration should be resorted to. If the patient recovers from the coma, the gastritis should receive appropriate treatment.

Postmortem Appearance.—When death has resulted from the inhalation of chloroform, no characteristic changes are found. Congestion of the lungs, bronchi, and kidneys has been described. The spleen has been found ruptured, but this seems to have been due to manipulations used in endeavoring to resuscitate the patient. The organs occasionally have the odor of chloroform. In cases of poisoning by liquid chloroform there are usually redness of the mucous membrane of the stomach and throat and sometimes ulceration. The epithelium of the

[1] See Case 4. [2] Marshall, *Medical News*, 1898, vol. lxxiii., p. 654.

[3] See, *e. g.*, Brouardel, *Bull. de l'Acad. de Méd.*, Feb. 1902. According to Embley (*loc. cit.*), of 83 cases of death which occurred in England during 1899, 68 happened before the operation was started; in another year it was 39 out of 41.

pharynx, glottis, and gullet may be softened and easily detached. If death has occurred in a short time, chloroform is found in the stomach; the contents of the stomach may have the odor of the poison. The body may resist putrefaction for some time. If death has been delayed for a few days, jaundice and fatty degeneration of the heart, liver, and other organs may be found; the latter has been observed also after prolonged narcosis and in cases of chronic poisoning.

CASES OF POISONING BY CHLOROFORM.

Eliot gives a summary of 57 cases of chloroform poisoning in which the drug was taken internally;[1] Hirsch gives a summary of 17 such cases.[2]

CASE 1.—Man, aged forty-three, swallowed at 7 A. M., after a sleepless night, about 1 ounce (45 gm.) of chloroform to secure sleep. When seen at 10.15 he was dazed, but on being roused could answer questions intelligently; breath smelt of chloroform. Complained of severe pain in umbilical region; constant desire to defecate and much straining. Had vomited once. Tongue and back of throat red and very sore. Fell asleep for a few minutes at a time; was awakened by pain. Pulse 120; respiration 56. At 12.20 P. M. began to pass blood from bowels; vomiting became frequent, and the vomited matter was blood-stained. No urine had been passed, although frequent painful attempts had been made; catheterized; urine was blood-stained. The patient remained awake and suffered intense pain; grew worse and died about 7.30 P. M., about twelve hours after taking the poison. No autopsy.[3]

CASE 2.—Patient very anemic; dreaded chloroform. Twenty or thirty drops given on lint, but before he was nearly under the influence and only a few seconds after he had been talking it was noticed that the breathing had become shallow; the pulse was fair and the reflexes were present. Conditions rapidly became dangerous; efforts to revive him failed. Patient was at no time under the influence of the anesthetic, and death was attributed to syncope caused by fear.[4]

CASE 3.—Child, aged five, anesthetized with "A. C. E. [alcohol, chloroform, and ether] mixture," anesthesia being continued by chloroform alone. Tonsils removed; pulse and respiration good. Signs of "coming round," so a little more chloroform was given preparatory to the removal of adenoids.[5] "She had hardly taken one or even two shallow breaths when her eyes suddenly became fixed, respiration ceased, and the pulse stopped." Child was inverted and artificial respiration maintained for twenty-five minutes, during which time there were two spasmodic gasps at an interval of a few minutes. Two drams (7.5 c.c.) of A. C. E. and 1 dram (3.7 c.c.) of chloroform had been used; much of latter had been wasted. Autopsy negative.[6]

CASE 4.—Man, aged forty-six, drank 5 ounces (222 gm.) of chloroform with suicidal intent—time not stated; vomited soon afterward. When brought to hospital seemed almost dead; no respiratory effort was visible, and the pulse could be felt only with difficulty. Face pale; lips cyanotic. Pupils widely dilated. Corneal reflex absent. After a few minutes of artificial respiration patient began to breathe. Stomach washed out; contents had no odor of chloroform, but the expired air did. Strychnin injected. Consciousness returned four hours after beginning of treatment. Complained of pain in mouth and abdomen. On the following day a scarlatiniform eruption on arms and legs. Left hospital after two days apparently entirely well.[7]

Isolation.—The isolation of chloroform from the tissues is accomplished by distillation with steam, and the apparatus described under

[1] *Medical Record*, 1885, vol. xxvii., p. 29.

[2] *Zeitschr. f. klin. Med.*, 1894, vol. xxiv., p. 190.

[3] Bridgman, *Lancet*, 1897, vol. ii., p. 384. [4] *Ibid.*, 1891, vol. iii., p. 1117.

[5] Death from chloroform seems to be especially frequent during operations for the removal of naso-pharyngeal adenoids.

[6] *Brit. Med. Jour.*, 1898, vol. i., p. 368.

[7] Dun, *Glasgow Med. Jour.*, 1898, vol. xlix., p. 347.

alcohol may serve here also, but the receiver should be surrounded with ice and provided with a mercury valve. As chloroform passes quickly into the circulation, an examination of the blood is of the greatest importance, and the acidity of the material under examination should be carefully noted. In case the reaction is neutral, a trace of tartaric acid should be added; if alkaline, a special examination must be made for formic acid, otherwise it will be impossible to say whether poisoning had been accomplished with chloroform or chloral, since the latter is decomposed by alkalis with the formation of chloroform[1]—

$$CCl_3CHO.H_2O + NaOH = HCO_2Na + H_2O + CHCl_3.$$

The slight solubility of chloroform in water also should not be neglected, so that even when no oil-drops appear, the aqueous distillate should, nevertheless, be submitted to the more sensitive tests for chloroform.

Tests.—1. On boiling a drop of chloroform with a mixture of 0.3 gram of resorcin in 3 c.c. of water and three drops of 10 per cent. sodium hydroxid, the fluid becomes yellowish-red and shows a green-yellow fluorescence. The red color is probably due to sodium rosolate and the fluorescence to fluorescein. A slight modification of the reaction may be applied directly to the urine.[2]

2. Chloroform precipitates red cuprous oxid when boiled with Fehling's solution. This reaction may be utilized for the quantitative determination of the poison. A measured amount of the reagent is heated in a pressure-bottle with the material in which chloroform is to be determined, and the precipitated cuprous oxid is reduced to copper in a stream of hydrogen and weighed; or the amount of unchanged copper may be determined with a standard solution of dextrose. Two equivalents of copper (2Cu) correspond to one equivalent of chloroform ($CHCl_3$):

$$CHCl_3 + 5KOH + 2CuO = Cu_2O + 3KCl + K_2CO_3 + 3H_2O.$$

3. On warming a mere trace of chloroform with a little anilin and alcoholic potassium hydroxid, the offensive odor of isobenzonitrile is produced. The test will easily show the presence of 1 part of chloroform in 5000 parts of water:

$$CHCl_3 + C_6H_5NH_2 + 3KOH = C_6H_5NC + 3KCl + 3H_2O.$$

This reaction is given also by chloral, bromoform, iodoform, and trichloracetic acid.

4. When warmed with *α*- or *β*-naphtol in strong caustic potash, chloroform produces a blue color, which, on exposure to the air, becomes green and finally brown.[3]

5. On boiling with alcoholic potassium hydroxid, chloroform yields the triethyl ether of orthoformic acid:

$$3C_2H_5OH + 3KOH + CHCl_3 = 3KCl + CH(OC_2H_5)_3 + 3H_2O.$$

[1] Liebig, *Liebig's Annalen*, vol. i., p. 199.
[2] Schwartz, *Zeitschr. f. anal. Chem.*, vol. xxvii., p. 668.
[3] Lustgarten, *ibid.*, vol. xxii., p. 97; *Monatsh. f. Chemie*, vol. iii., p. 722.

After diluting with water and evaporating the alcohol, the material is acidified with tartaric acid and the formic acid distilled off and tested by reactions given on page 553 under Chloral.

6. A test for chloroform which renders others almost superfluous is founded on the decomposition of the substance by heat into perchlorbenzene, chlorin, and hydrochloric acid.[1] This reaction forms the basis of the Ragsky[2] process, which should always be employed where circumstances point to chloroform, and especially when the amount of the poison (as in an examination of the blood) is known to be small. The material is introduced into a flask provided with a doubly perforated cork. Through one of the perforations passes a funnel tube, and through the other a delivery tube, bent at right angles. The flask is placed on a water-bath and connected with a piece of hard glass tubing eighteen inches long, which is heated for about four inches by a broad Bunsen burner. About four inches in front of the heated portion the tube

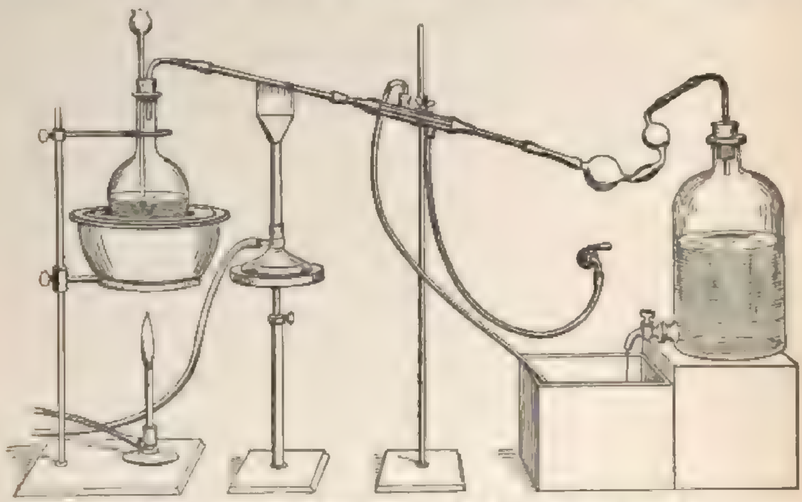


FIG. 62.—Apparatus for detecting chloroform by the Ragsky process.

passes through a Liebig's condenser having a length of six inches, and beyond the condenser a piece of filter-paper, moistened with a mixture of starch-paste and potassium iodid, is placed in the tube. The end of the tube is connected with a set of Liebig's bulbs containing a solution of silver nitrate, and these are in turn connected with an aspirator.

After the tube has been heated to bright redness the mixture in the flask is distilled at a gentle heat, while a current of air is slowly drawn through the system by the aspirator. Any chloroform vapor that may be present is decomposed for the most part according to the equation:

$$6CHCl_3 = C_6Cl_6 + 6HCl + 6Cl.^3$$

[1] Basset, *Jahresb. d. Chemie*, 1867, p. 608; Ramsay, *ibid.*, 1886, p. 628.

[2] *Jour. f. prak. Chem.*, vol. xlvi., p. 170.

[3] At a dull red heat monochlorethane and tetrachlorethylene are formed at the expense of perchlorbenzene. See Ramsay and Young, *Jahresb. u. die Forts. der Chemie*, 1886, p. 628.

With 150 c.c. of blood taken from the carotid of a dog anesthetized with chloroform the writers were able to show all three products with the greatest ease. The perchlorbenzene forms beautiful needles just in front of the flame; the chlorin decomposes the potassium iodid, liberating iodin, which turns the starch blue, and hydrochloric acid, passing into the bulbs, precipitates silver chlorid, which may be identified by appropriate tests.

If the presence of chloroform is thus indicated, the heating of the tube should be discontinued and the distillate collected in a pointed tube. Any oil-drops which may thus be collected should be submitted to the tests given above.

In the Maxwell case the presence of chloroform was demonstrated by Luedeking[1] in the body of the victim twelve days after death from the inhalation of chloroform, and in the bodies of dogs killed in this manner after they had been permitted to decompose for four weeks. Maxwell afterward confessed that he had given chloroform. Luedeking used the lungs for analysis, and cites as a partial explanation of the tenacity with which chloroform is held by the tissues that a solution of chloroform water will respond without difficulty to the tests for chloroform after it has been exposed to the air for two weeks.

Animals dying from other causes and left to decompose for ten days to four weeks gave no reaction by the Ragsky process simulating that produced by chloroform.

BROMOFORM.

Bromoform ($CHBr_3$) is a heavy, oily liquid very similar to chloroform. The vapor produces anesthesia, but is very irritating to the eyes and respiratory passages; it is seldom used as an anesthetic. Bromoform in the liquid form, administered in an aqueous vehicle, has been used quite extensively recently in whooping-cough. A number of cases of poisoning, for the most part not fatal, have been reported. In many of these cases poisoning resulted from the administration of the last dose in the bottle; the heavy bromoform had sunk to the bottom, and an overdose was thus administered.[2] The symptoms are very similar to those caused by chloroform taken internally; there are, first, excitement and intoxication, then a tendency to sleep, anesthesia, coma, and collapse. Three drops are said to have caused serious symptoms in a child of four; a child of the same age was severely, but not fatally, poisoned by 15 or 20 minims (2.6 or 3.5 gm.).[3] About 1 dram (3.75 c.c.) caused the death of a child of two years in five hours;[4] convulsions occurred in this case.

Long-continued administration of bromoform to animals causes fatty degeneration of various organs.

Detection.—Bromoform may be isolated and identified by methods

[1] *Amer. Chem. Jour.*, 1886, p. 358.

[2] Cheney, *Arch. of Pediatrics*, 1887, vol. xiv., p. 112; Burton-Fanning, *Brit. Med. Jour.*, 1901, vol. i., p. 1202.

[3] Dean, *Lancet*, 1893, vol. i., p. 1062.

[4] Müller, *Munch. med. Wochenschr.*, vol. lv., p. 1211.

which are analogous to those given for chloroform. It shares with chloroform the isobenzonitrile reaction, but differs from this substance in its failure to produce a formate when boiled with alcoholic potassium hydroxid :[1]

$$CHBr_3 + 3KOH + C_2H_6O = 3KBr + CO + C_2H_4 + 3H_2O.$$

IODOFORM.

Most of the cases of poisoning with iodoform have been medicinal and have resulted from the too free use of the drug as a surgical dressing ; a few cases have resulted from its internal administration. Poisoning with iodoform is most frequent in persons after middle life.

Properties.—Iodoform (CHI_3) forms small lemon-colored crystals possessing a very penetrating, persistent, and disagreeable odor and taste ; it is insoluble in water and soluble in alcohol and ether. It melts at 119° C. (246.2° F.) and is volatile with steam.

The **physiologic action** of iodoform is exceedingly complex. Part of the drug is absorbed as such and causes marked effects upon the brain, while part is decomposed by alkalis and proteids into iodin and iodids, and these exert their peculiar action upon various tissues. Moreover, the secretion of the thyroid gland is thought to be increased, and the acceleration of the heart, which is so often observed in cases of poisoning with iodoform, is frequently attributed to this action. The local action of iodoform is not, as a rule, very marked, although at times it causes irritation in the neighborhood of the wound. The iodin of iodoform is chiefly excreted in the urine as iodids, but some is found in the saliva, sweat, and bronchial secretion. Some of the iodin is retained in the body for a very long time, and is excreted in the urine very slowly.

Symptoms.[2]—The symptoms of acute iodoform poisoning, as might be expected from what has been said above, are very complex and variable, and sometimes one symptom and sometimes another predominates. As seen after the free application of iodoform to a wounded surface, they are frequently as follows : There may be general malaise for a few hours or a day, and then the more severe symptoms appear. The patient becomes sleepless and restless, may suffer from nausea, headache, giddiness, and mental confusion, and then pass into a condition of great depression resembling melancholia ; hallucinations and delusions of persecution and suicidal impulses may follow. The period of depression may be followed by mania and violent delirium and other symptoms resembling meningitis. Coma, often continuing for several days, and finally collapse and death occur. At times the immediate effects disappear, but permanent insanity and dementia result. In other cases the delirium is of a mild type, and the delusions harmless but grotesque.[3]

The pulse in cases of iodoform poisoning is often very rapid. There

[1] *Liebig's Annalen*, vol. cxciv., p. 23.
[2] See McLean, *Amer. Gyn. and Obstet. Jour.*, 1897, vol. x., p. 249.
[3] Black, *Brit. Med. Jour.*, 1885, vol. i., p. 70.

is often high fever, but this may not be present. In some cases the symptoms of cerebral irritation are absent, the symptoms beginning with somnolence which deepens into stupor and collapse.

All the foregoing symptoms may occur after the removal of the dressings. The most frequent form of poisoning is that in which the effects are largely local. In these cases, which are frequently ascribed to an "idiosyncrasy," there are redness and painful swelling about the wound, and often a wide-spread eruption and lymphangitis and sometimes death—in one case after twenty days.

The long-continued use of iodoform in wounds leads to a chronic form of poisoning in which the symptoms of cerebral excitement are but little marked. Dyspepsia and loss of weight, eczema, and other skin eruptions, palpitation of the heart and amblyopia [1] are frequent features of this form of poisoning.

Fatal Period.—The amount of iodoform necessary to cause death when taken internally is not known; individuals seem to differ greatly in their susceptibility to the drug. Thirty grains (1.94 gm.) and perhaps even 20 grains (1.29 gm.) have caused death, while recovery has followed the taking of 120 grains (7.77 gm.). Very severe symptoms have followed the application of 11 grains (0.71 gm.) of iodoform to the uterus,[2] but such a result must be regarded as extremely unusual. The poisonous effects caused by the absorption of iodoform from wounds depend, not upon the amount of iodoform in the wound, but upon the amount actually absorbed, and the conditions determining the absorption are not known. Death may occur in a few days, or it may be delayed for weeks.

Treatment.—The dressings should be promptly removed upon the appearance of symptoms of poisoning, but even then they may continue or become more severe. Potassium bromid has been highly recommended. In grave cases intravenous or subcutaneous infusions of normal saline solutions are said to have given good results. In the severe cerebral cases the prognosis is bad.

Postmortem Appearances.—Fatty degeneration of the heart, liver, and kidneys is the most characteristic lesion. Edema of the pia mater and of the lungs and acute nephritis have been found in some cases.

Detection.—The material under examination is made alkaline with caustic potash and submitted to distillation in steam. The distillate is treated with a few drops of caustic potash, and any iodoform present is shaken out with ether in a separating funnel. After the ether evaporates, which should occur at the room-temperature, the iodoform remains as hexagonal stars or other hexagonal forms which may be recognized under the microscope (see Fig. 61).

The odor of iodoform is very characteristic, but unfortunately this odor is often masked by other odors which are present in a distillate from an organic fluid. Lustgarten,[3] therefore, suggests the following

[1] *Therap. Gaz.*, 1897, p. 671. [2] *Ibid.*, 1888, p. 188.
[3] *Monatsh. f. Chemie*, vol. iii., p. 717.

reaction, which will show the presence of a small fraction of a milligram of the substance:

1. To a portion of a solution of 20 parts of phenol or resorcin and 40 parts of sodium hydroxid in 70 parts of water are added a few drops of a solution of the suspected substance in alcohol, and the mixture is carefully warmed over a small flame. In the presence of iodoform a red precipitate forms, which is soluble in alcohol with a carmin-red color. The red color is discharged by mineral acids and restored by alkalis. This red substance has all the properties of rosolic acid, and is produced by chloroform as well as by iodoform.

2. Iodoform also yields the odor of isobenzonitrile when warmed with anilin and alcoholic potassium hydroxid.

3. When the material is obtained in sufficient quantity, a portion may be fused with caustic potash and tested for iodin.

SULPHONAL.

Sulphonal was introduced into medical practice in 1888, and within six years had caused at least eighteen deaths,[1] most of which were medicinal or accidental.

Properties.—Sulphonal is the copyrighted name of diethyl-sulphone-dimethyl-methane—$(CH_3)_2C(SO_2C_2H_5)_2$. It occurs in colorless crystals that are without odor and nearly tasteless. They melt at 125° C. (257° F.). Sulphonal is sparingly soluble in cold, readily soluble in hot, water, and soluble also in alcohol and ether.

The **physiologic action** of medicinal doses of sulphonal is very nearly the same as that of paraldehyd—*i. e.*, it causes sleep, with but little depression of the circulation and respiration. Its insolubility in water, however, causes the absorption to be irregular, and hence the effect to be somewhat more uncertain than that of paraldehyd. Sulphonal is eliminated by the kidneys partly as such, partly as ethylsulphonic acid; the elimination takes place very slowly, so that there is a tendency to a cumulative action.

Symptoms.—Medicinal doses—15 to 30 grains (0.97 to 1.94 gm.)—of sulphonal usually simply cause sleep, but sometimes nausea, headache, dizziness, and irregular gait result. After larger doses the symptoms are exceedingly variable; among those noted are the following: mental confusion; motor disturbances, as shown by an irregular gait, stupor, and insensibility; more rarely excitement and convulsions. In very severe cases the respiration is stertorous and irregular, the pulse scarcely perceptible, and there is marked cyanosis. There is sometimes some elevation of temperature. Death usually results from failure of the respiration, but it may occur although artificial respiration is maintained. Sometimes the secretion of urine is suppressed and death results from anuria. A very common feature, especially in those cases which continue for some time, is the appearance of hematoporphyrin in the urine;[2] this pigment, which indicates an extensive destruction of

[1] Friedlander, *Therap. Monatsh.*, 1894, p. 281.

[2] See Tyson-Croftan, *Phila. Med. Jour.*, 1902, vol. ix., p. 882.

the blood, causes the urine to assume a red color and is an unfavorable symptom. Nearly all the cases of hematoporphyrinuria reported have occurred in anemic women. Skin eruptions may also follow a single large dose. Coma has continued for six days and recovery taken place.

The long-continued use of sulphonal leads to a form of chronic poisoning[1] in which headache, vomiting, constipation, albuminuria, and hematoporphyrinuria, various mental and motor disturbances, and various forms of skin eruptions have been described.

Fatal Dose.—The fatal dose is extremely variable—much depends upon the individual. Thirty grains (1.94 gm.) caused death in a neurasthenic woman in forty hours.[2] Death has frequently followed doses varying from 75 grains (4.85 gm.) to 1 ounce (31 gm.). On the other hand, a boy of fifteen took over 3 ounces (93 gm.), and after sleeping ninety hours recovered.[3] In many cases death has resulted after the long-continued use of comparatively small doses. Thus several deaths have followed the daily use of from 10 to 20 grains (0.64 to 1.29 gm.) for from two to twelve months.[4]

Fatal Period.—Death may occur in a few hours or days[5] or after months; it has occurred some time after the use of the drug was stopped.

Treatment.—The stomach should be washed out, and diuretics, purgatives, strychnin, and other respiratory stimulants administered. Much may be done to avert the poisonous symptoms, so often observed when the drug is used for some time, by observing certain simple precautions. The drug should be administered in solution in hot water, in order to secure more rapid and complete absorption; the administration should be interrupted every few days. These precautions greatly diminish the tendency to the appearance of a cumulative action.

Case of Sulphonal Poisoning.—A woman, aged twenty-eight, suffering from melancholia and hysteric manifestations, but apparently free from organic disease, took 15 grains (0.97 gm.) of sulphonal; as wakefulness continued another dose of 15 grains (0.97 gm.) was given an hour and a quarter later. She slept for twelve hours; she could then be roused and would talk rationally, but when left alone, would fall asleep again. The temperature rose to 102° F. Respiration began to fail, and the patient became very cyanotic after about twenty-three hours; artificial respiration was maintained for several hours, the pulse continuing fairly good for some time. Death occurred forty hours after taking the drug.[6]

Isolation.—Sulphonal is one of the most stable substances known to chemistry. It dissolves in concentrated sulphuric acid, may be recovered unchanged by diluting the solution with water. Boiling alkalis and concentrated nitric acid are likewise without effect upon the substance.[7] This chemical stability prevents the decomposition of sulphonal in the body after death, and is, therefore, favorable to its postmortem detection. The substance is isolated from organic matter by extraction

[1] Pollitz, *Wien. klin. Wochenschr.*, 1898, vol. ii., p. 566.
[2] Case 1.
[3] Neisser, *Deutsch. med. Wochenschr.*, 1891, vol. xvii., p. 702.
[4] Pollitz, *loc. cit.*
[5] Knaggs, *Brit. Med. Jour.*, 1890, vol. ii., p. 955.
[6] Pettit, *Medical News*, 1889, vol. lv., p. 165.
[7] Baumann, *Ber. d. d. chem. Ges.*, vol. xix., p. 2808.

with alcohol, evaporation of the alcoholic fluid, extraction of the residue with hot water, evaporation of the aqueous liquid, and final extraction of the residue with ether. If the residue obtained after evaporating the ether is large, it may be recrystallized from hot water[1] and its melting-point determined in addition to chemical tests; otherwise the tests should be applied directly.

The following method is especially applicable to the urine. A liter or more of urine is evaporated to 100 c.c. and shaken out six times with 200 c.c. of ether which contains a little alcohol. The united extracts are evaporated to dryness, the residue taken up in 20 c.c. of 10 per cent. caustic soda and again evaporated to dryness. This residue is taken up in 20 to 40 c.c. of water, and the aqueous fluid extracted repeatedly with ether, which is allowed to stand overnight and filtered through a washed filter to remove any traces of water. On evaporation of the ether sulphonal will remain pure and in crystalline form.[2]

Tests.—1. When melted with potassium cyanid sulphonal develops an offensive mercaptan odor, and potassium sulphocyanate is formed at the same time. A blood-red color is, therefore, produced on the addition of ferric chlorid to a solution of the residue in water.[3] A careless execution of this test is somewhat dangerous to the experimenter.

2. When heated in a test-tube with powdered charcoal, sulphonal forms mercaptan, acetic acid, formic acid, and sulphur dioxid. The odor of mercaptan may be noted, and the vapors will change blue litmus-paper. Sulphur dioxid may be shown by its bleaching action on a piece of filter-paper moistened with blue starch iodid and suspended in the mouth of the tube.[4]

TRIONAL AND TETRONAL.

Trional is sulphonal in which one of the methyl groups has been replaced by an ethyl (C_2H_5) group; in tetronal both of the methyl groups have been replaced by ethyl. These compounds are very similar in both their chemical and physiologic properties to sulphonal,[5] but both have a bitter taste, and trional melts at 76° C. (169° F.) and tetronal at 89° C. (192° F.). Both compounds have caused fatal poisoning; the symptoms are very similar to those caused by sulphonal.[6]

ORGANIC ACIDS OF THE FATTY ACID SERIES.

Poisoning with these acids is extremely rare, yet death has been caused by large amounts of acetic and tartaric acids. The symptoms are very similar to those caused by the mineral acids (*q. v.*). Stumpf[7]

[1] Sulphonal is soluble in 15 parts of boiling water and 500 parts of water at 15° C.
[2] Morro, *Deutsch. med. Wochenschr.*, 1892, p. 672
[3] Vulpius, *Zeitschr. f. anal. Chem.*, vol. xxvii., p. 665.
[4] Schwartz, *ibid.*, vol. xxvii., p. 665.
[5] Baumann and Kast, *Zeitschr. f. physiol. Chem.*, vol. xiv., pp. 63, 64.
[6] Several cases are cited by Church, *Amer. Med.*, vol. ii., p. 729; see also Young, *Univ. Med. Mag.*, vol. ix., p. 715.
[7] Stumpf, *Munch. med. Wochenschr.*, 1898, vol. xlv., p. 690.

has reported a fatal case of poisoning by a tablespoonful of the "essence of vinegar" (pure acetic acid); the man, aged thirty-two, suffered from severe diarrhea and a weak heart. Death occurred on the third day; for two days before death the patient had been in a condition of stupor. In a case reported by Birkett[1] 2 or 3 ounces (59.2 or 89 c.c.) of 33 per cent. acetic acid were swallowed by an intoxicated man; in addition to the severe gastric symptoms there was laryngeal obstruction necessitating tracheotomy. Recovery followed.

Tartaric acid is sometimes used as an ingredient of lemonade; very serious poisoning has resulted from neglect properly to dilute the mixture.

ETHER.

Ether has but little toxicologic importance. The most frequent cause of death from ether has been its use as an anesthetic; it is estimated that one death occurs in about 12,000 etherizations. It has also been used as a means of committing suicide, and chronic poisoning has resulted from its use as an intoxicant.

Properties.—Ether (*Æther*, U. S. P., sulphuric ether, ethyl oxid, $(C_2H_5)_2O$) is a colorless liquid of peculiar penetrating odor and sweetish, pungent taste. It evaporates very rapidly, producing great cold. Pure ether has a specific gravity of 0.713 and a boiling-point of 35° C. (95° F.), but commercial ether usually has a higher specific gravity and a higher boiling-point, for it contains alcohol (usually about 4 per cent.) and a little water. Ether is very inflammable and its vapor forms a highly explosive mixture with air. Ether is easily soluble in chloroform and alcohol, sparingly so in water.

The physiologic action of ether is, in the main, similar to that of alcohol, but it acts more promptly and the effects pass off more rapidly.

Symptoms.—The symptoms following the inhalation of ether are very similar to those due to chloroform, as already described. The vapor is irritating to the larynx and gives rise to a feeling of suffocation; the respiration and heart are slowed reflexly and rendered irregular. The secretion of mucus and of saliva is much increased. Consciousness and sensation are soon lost, and if too much ether is given, death results—usually from failure of the respiration.[2]

Ether is more irritating to the lungs and perhaps to the kidneys than is chloroform; pneumonia sometimes follows its administration.[3]

When ether is swallowed there are an intense burning sensation in the throat and stomach and a rapid, intense intoxication, similar to but of shorter duration than that caused by alcohol. The irritation may lead to inflammation of the stomach and intestines. Individuals may become habituated to the use of ether just as to alcohol, and be able to take large amounts without marked immediate effects; such a practice leads, however, to many nervous symptoms, such as trembling of the

[1] Birkett, *Lancet*, 1867, vol. ii., p. 98.

[2] See, *e. g.*, case reported by Biddlecombe, *Brit. Med. Jour.*, 1892, vol. i., p. 437; Herhold, *Berlin. klin. Wochenschr.*, 1894, vol. xxxi., p. 589.

[3] See Crouch and Corner, *Lancet*, 1902, vol. i., p. 1457.

hands, muscular weakness, cramps of the muscles, headaches, etc. The practice of ether-drinking is quite common in parts of Ireland.[1] Occasionally a person acquires the habit of inhaling ether in order to secure the intoxicating effects.

Fatal Dose.—The fatal dose of ether when inhaled varies within very wide limits; it is largely a matter of the concentration of the vapor. Spenzer[2] found that air containing 6 per cent. of ether vapor caused stoppage of the respiration in ten minutes, while 3.5 per cent. was sufficient to produce narcosis. It is stated that death has resulted from the inhalation of quantities varying from 2½ to 16 ounces (75 to 480 c.c.). The fatal dose when taken internally is not known; an individual has recovered from 7 drams (26 c.c.). It is thought that 1 fluidounce (30 c.c.) would be fatal to most adults. Ether-takers become accustomed to large amounts; there is a case reported in which a boy gradually learned to consume 2 pints (946 c.c.) of ether daily, taking part by inhalation, part internally.[3]

Fatal Period.—Death may occur at any stage of ether narcosis

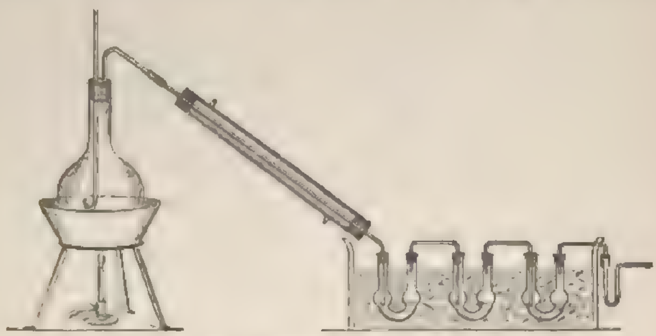


FIG. 63.—Apparatus for detecting ether

or before narcosis has been produced; or it may take place hours or days later from pneumonia or inflammation of the kidneys.

Treatment.—In cases of arrest of breathing during ether anesthesia artificial respiration should be promptly practised. Strychnin, ammonia, or digitalis may be given hypodermically as stimulants. The inhalation of oxygen is also recommended.

Postmortem appearances are not characteristic. There is usually hyperemia of the mucous membranes, and if the autopsy be made soon after death, the organs have an odor of ether.

Detection.—Owing to the great volatility of ether, its isolation from animal tissues is attended with some difficulty; in fact, the poison is likely to escape detection unless the attention of the analyst is specially directed to the substance by attendant circumstances.

A form of apparatus that is suitable for the distillation consists of

[1] Hart, *Brit. Med. Jour.*, 1890, vol. ii., p. 885.
[2] Spenzer, *Archiv f. exper. Path. u. Pharmakol.*, 1894, vol. xxxiii., p. 407.
[3] Sedant, *Gaz. des Hôp.*, 1883, p. 814.

an ordinary distilling flask, into which the material is to be introduced, provided with a cork, through which passes a long open glass tube drawn out at its lower end and bent upward. This tube should scarcely dip below the surface of the liquid in the flask, and serves to admit air at the end of the operation. The exit-tube of the flask enters a Liebig's condenser which is connected by means of an adaptor with a succession of three Pelligot tubes and a final U-tube containing a little mercury. All connections should be made with well-fitting corks (not rubber stoppers) and the system tested with a filter-pump (see Fig. 63).

The material under investigation is placed in the flask, the Pelligot tubes surrounded with a mixture of ice and salt, and the amount of mercury in the valve so arranged that inward pressure will be relieved by the entrance of air into the flask through the long tube. Distillation is then carried on very slowly by means of a warm water-bath. The distillate, if obtained in quantity, is treated with fused calcium chlorid and submitted to a second distillation with all the precautions noted; but, ordinarily, the amount of ether recovered will be limited to a few drops that will be found in the first Pelligot tube. The tube is disconnected and closed with solid corks, one of which is provided with a slit carrying a small piece of filter-paper that has been moistened with a mixture of sulphuric acid and bichromate of potassium. In the presence of ether vapor the bichromate will be reduced to green chromium sulphate. This reaction in connection with the properties already noted (volatility, odor, combustibility) sufficiently identifies the substance.

AMYL NITRITE.

Amyl nitrite ($C_5H_{11}NO_2$) is a very volatile liquid with a peculiar, fruit-like odor. Its specific gravity is 0.877, and the boiling-point, 96° C. (204.8° F.). It is insoluble in water. It is given in medicine by inhalation, to cause dilatation of the arteries. The chief symptoms are flushing of the face and upper parts of the body. After large doses vomiting, unconsciousness, and collapse, with shallow respiration and cyanosis, may occur. Curious disturbances of vision also result. In the lower animals amyl nitrite causes the blood to assume a chocolate color, due to the formation of methemoglobin. The symptoms following the internal administration of amyl nitrite are similar to those caused by the inhalation of the drug. In one case a man,[1] aged sixty, took a teaspoonful; the chief symptoms were great weakness, cyanosis, and a very feeble, slow, intermittent pulse. Recovery occurred under appropriate treatment (emetics, strychnin, and digitalis).

Detection.—Upon distillation of the material under examination amyl nitrite will be found in the distillate, from which it may be separated by agitation with ether. In spite of its high boiling-point, amyl nitrite evaporates rapidly at the ordinary temperature, so that special precaution is required to prevent its loss during the distillation. On heating the aqueous distillate with caustic potash the substance

[1] Shoemaker, *Medical News*, vol. lxii., p. 544.

decomposes with the formation of amyl-alcohol and potassium nitrite. The latter can be shown by any of the large number of tests, such as the yellow or brown color that it imparts to an acidified solution of metaphenylenediamin.

ETHYL BROMID.

Ethyl bromid (hydrobromic ether, C_2H_5Br) is a heavy, colorless, very volatile liquid, with a chloroform-like odor. When inhaled, it produces anesthesia with great rapidity. Consciousness returns within two or three minutes after the administration is discontinued. The rapidity with which the effects appear and pass off again has led to the somewhat extensive use of ethyl bromid for brief surgical operations, as in dentistry. Ten volume per cent. is necessary to anesthetize animals within five minutes, and if this concentration is maintained for fifteen minutes, death occurs: hence it cannot be considered as an entirely safe anesthetic. As a matter of fact, a number of deaths have occurred from its use. In a series of 60,000 cases there were 16 deaths—*i. e.*, one death in 3750. In another series there was one death in 5220 cases. In some of these cases death resulted from the long-continued administration of the anesthetic, a practice that does not seem to be justified. Death may occur at any period of the administration, as within thirty seconds after the beginning [1] or hours [2] and days afterward.

Ethyl bromid readily undergoes decomposition; this change is indicated by the appearance of a yellow color. Such a preparation is very poisonous, and some deaths have very probably been due to its employment. Death has also resulted from the use of ethylene bromid ($C_2H_4Br_2$), this substance, which is much more poisonous than ethyl bromid, having been dispensed instead of ethyl bromid.[3] Several deaths have also resulted from the employment of ethyl bromid first and then of chloroform; in one of these cases death occurred seven days afterward, with the symptoms of acute yellow atrophy of the liver.[4] About $\frac{1}{4}$ of an ounce of ethyl bromid is said to have caused the death of an anemic and hysteric woman; in other cases the fatal dose has been from 1 to 2 ounces.

Detection.—Ethyl bromid is sufficiently distinguished from other poisons containing bromin by its low boiling-point, but, owing to its instability, it is not likely to be recovered from the tissues. Ethylene bromid decomposes quantitatively when heated with zinc and a little water, producing zinc bromid and ethylene gas—[5]

$$C_2H_4Br_2 + Zn = Zn\,Br_2 + C_2H_4.$$

The reaction may be performed with a very small quantity of material, and the gas shown to burn with a smoky flame.

[1] *Phila. Med. Jour.*, 1899, vol. iv., p. 367 [2] Sims, *Medical Record*, 1880, p. 361.
[3] Scherbatscheff, *Arch. f. exper. Path. u. Pharmakol.*, vol. xlvii., p. 1. See also *Therap. Monatsh.*, 1888, p. 556; 1889, p. 385.
[4] Reich, *ibid.*, 1893, p. 250.
[5] Gladstone and Tribe, *Ber. d. d. chem. Ges.*, vol. vii., p. 365.

NITROGLYCERIN.

Many of the cases of poisoning by nitroglycerin are "industrial," and occur among workmen engaged in its manufacture or in the manufacture and use of dynamite and other high explosives; other cases are due to accident or to its use for the purposes of murder. In most of the cases of criminal poisoning the nitroglycerin has been added to alcoholic drinks. Some interesting testimony as to the use of nitroglycerin was given a few years ago by a criminal.[1] He said: "The way to work it was to engage a boat on a warm day, entice the victim to go for a row, give him a drink of whisky dosed with nitroglycerin, and then set him adrift; when discovered dead, they would pronounce it a case of death from sunstroke." He had practised the use of different poisons on poor tramps in the slums of Chicago, and the nitroglycerin "was the surest and the best." He also said that a chemist could easily be deceived by it.

Properties.—Nitroglycerin (glyceryl trinitrate, or glonoin, $C_3H_5(NO_3)_3$) is a pale yellow, oily liquid that is nearly insoluble in water, soluble in absolute alcohol, ether, and chloroform. It can be ignited in an open vessel, when it may burn quietly; but when heated above 250° C. (482° F.), it explodes. Similar violent explosions are brought about by concussion. A 1 per cent. solution of nitroglycerin in alcohol is used in medicine under the name of *Spiritus Glonoini* (U. S. P.).

The **physiologic action** of nitroglycerin is similar to that of amyl nitrite, but its effect is more prolonged. It is an extremely active drug, the medicinal dose being from $\frac{1}{2}$ to 2 minims (0.03 to 0.1 c.c.) of the 1 per cent. solution; this corresponds to from $\frac{1}{200}$ to $\frac{1}{50}$ of a minim (0.0003 to 0.001 c.c.) of nitroglycerin. One or 2 minims (0.05 to 0.1 c.c.) of the 1 per cent. solution may cause such powerful pulsations that a pen held in the hand is visibly jerked.[2] The drug is absorbed by the unbroken skin, and, in the form of vapor, by the lungs, as well as from the mucous membranes.

Symptoms.—The most marked symptom in the milder cases of poisoning is a very severe headache—the "powder headache" of those who are engaged in the manufacture of nitroglycerin,[3] dynamite, ballastite, cordite, etc. The poisoning may result from the taking of the nitroglycerin internally or in the form of vapor, or it may be absorbed by the skin. A single drop rubbed into the skin may cause a headache lasting for ten hours.

When a large dose of nitroglycerin is taken internally, the chief symptoms are a burning sensation in the throat, vomiting, colicky pains, and sometimes diarrhea. The arteries of the head dilate and throb; severe headache is almost invariably present, and dizziness and weakness follow. The respiration is at first accelerated, but it soon becomes dyspneic; cyanosis is marked, and in prolonged cases some methemo-

[1] See *Boston Med. and Surg. Jour.*, vol. cxxxi., p. 458.
[2] Murrell, *Lancet*, 1879, vol. i., p. 80.
[3] *Jour. Amer. Med. Assoc.*, vol. xxxi., p. 798.

globin is probably formed. Coma develops, and death results from failure of the respiration. Delirium is sometimes observed.

Workmen in factories, as well as patients receiving the drug medicinally, rapidly acquire a tolerance for it; thus a case is reported in which a patient became accustomed, in the course of a year, to 6 grains (0.39 gm.) daily. There is, however, a chronic form of poisoning among those who handle nitroglycerin a great deal; among the effects are ulcers on the fingers and skin eruptions.

Fatal Dose and Period.—In most of the fatal cases a large amount of the poison has been taken, but it seems probable that a very few drops would prove fatal—$\frac{1}{60}$ of a minim (0.001 c.c.) has produced most severe headache. Even after large doses death does not seem to have occurred for at least two hours; in other cases it was delayed for six hours or more.

Treatment.—The elimination of the poison should be hastened by washing out the stomach and by the administration of cathartics. In severe cases blood-letting, with subsequent saline transfusion, may be resorted to. Large quantities of black coffee seem to give great relief to the severe headaches.

The chief **postmortem appearances** are hyperemia of the stomach and intestines and also of the brain and its membranes. The mucous membrane of the air-passages may be of a reddish-brown color.

Isolation.—Owing to the unstable character of nitroglycerin in the presence of reducing agents and the close correspondence of its solubilities with those of the fats, the isolation of the substance from the tissues is attended with considerable difficulty. Moreover, Werber's experiments upon poisoned animals show that the poison is rapidly decomposed in the body and is not likely to be found in the urine, blood, or liver. The attention of the analyst should, therefore, be specially directed to the stomach-contents, as well as to vomited matter, and any suspicious oil-drops found in the digestive tract should be submitted to tests before purification.

Werber's method[1] of extracting nitroglycerin from the organic matter by direct treatment with chloroform or ether will, in many instances, yield satisfactory results; but, owing to the great difficulty of removing the solvent after it has been thoroughly shaken with organic matter, the following procedure will often be found advisable. The material under examination is faintly acidified with sulphuric acid, and digested for twenty-four hours with five or six times its volume of absolute methyl-alcohol. The solution is then filtered, most of the alcohol is driven off on the water-bath, and the residual fluid, which should not be filtered, is shaken out with ether or chloroform. Upon evaporation of the organic solvent nitroglycerin will remain as a viscous oil. Although the substance will be highly contaminated with fat, it is well to make a preliminary test at this point, and especially when the amount of material is small. On treatment with a small quantity of cold alcohol the nitroglycerin will pass easily into solution, leaving the fat, and may

[1] *Zeitschr. f. anal. Chem.*, vol. vii., p. 158.

be thrown down as oil-drops by the addition of a sufficient amount of water.

Tests.—1. Nitroglycerin explodes violently when heated in a capillary tube.

2. A tenth of a milligram of nitroglycerin may be shown by the purple-red color that is produced upon treatment with a trace of anilin or brucin and a drop of concentrated sulphuric acid.[1] The test is given also by nitrates, nitrites, and many nitro-compounds.

3. The following reaction requires a great expenditure of material, but, in the absence of fats, furnishes valuable evidence in the identification of nitroglycerin. The substance is evaporated on the water-bath with a strong aqueous solution of yellow ammonium sulphid. Glycerin and ammonium nitrite are formed:[2]

$$C_3H_5(NO_3)_3 + 3NH_4HS = C_3H_5(OH)_3 + 3NH_4NO_2 + 3S.$$

The pasty residue is extracted with alcohol, which, upon evaporation, leaves the glycerin. This may be identified by its sweet taste and by the odor of acroleïn, which is produced when the substance is heated dry with acid potassium sulphate:

$$C_3H_5(OH)_3 = C_2H_3CHO + 2H_2O.$$

A second portion of the suspected substance is treated successively with yellow ammonium sulphid, an excess of lead carbonate, and a trace of lead acetate. After filtration the liquid is tested for a nitrite with metaphenylenediamin and sulphuric acid.

HYDROCYANIC ACID AND THE CYANIDS.

Hydrocyanic acid and some of its salts are used very extensively in the arts, and to a much more limited extent in medicine. They are readily accessible to the public. Hydrocyanic acid is also found very widely distributed in nature; it occurs, almost always in more or less firm combination, in many plants, and is contained in certain flavoring agents or liqueurs derived from such plants. It is also found in the secretions of a few myriapods.

The *cyanids* (hydrocyanic acid and its compounds) occupy a very prominent place in the list of poisons; in the statistics of some European countries they occupy the third or fourth place in the order of the frequency with which death is caused by poisons. In England alone, for the ten years ending in 1892, there were 426 deaths from the cyanids;[3] only the opiates, lead, and carbolic acid, among the poisons, were responsible for a greater mortality. Of 432 cases of poisoning reported in Berlin for the three years 1876–78, 40 were due to the cyanids.[4] Accurate statistics for the United States are difficult to obtain, but of 286 cases of poisoning collected for the years 1893 and 1894, there were but 11 cases due to the cyanids.

[1] Werber, *loc. cit.* [2] Bloxam, *Chemical News*, vol. xlvii., p. 169.
[3] Blyth, *Poisons*, 1895, p. 30.
[4] Lesser, *Virchow's Archiv*, 1881, vol. lxxxiii., p. 198.

The greater number of the cases of cyanid poisoning are due to suicide. Thus, of the 426 cases reported in England for the ten years ending in 1892, 344 cases were due to the use of the cyanids for suicidal purposes; 38 cases were due to accident or negligence, while in but 14 were they used for the purpose of murder. In 793 poisoning cases of a criminal character in France occurring in the twenty-one years 1851–71, the cyanids were used in but 4. Witthaus[1] was able to find in the literature up to 1896 but 31 cases in which hydrocyanic acid and its compounds were alleged to have been used for purposes of murder.

In some statistics the greater number of cases were due to aqueous solutions of hydrocyanic acid itself; in others, to potassium cyanid. The former has been used most frequently for suicidal purposes by pharmacists, chemists, and physicians; the latter has been used chiefly by photographers and electroplaters. Poisoning with the various substances containing hydrocyanic acid (oil of bitter almonds, peach-kernels, etc.) is not very common.

Properties.—Hydrocyanic acid (hydrogen cyanid or prussic acid, HCN) is a colorless, transparent liquid of a penetrating characteristic odor resembling that of peach-kernels. It boils at 26.5° C. (80° F.), and solidifies at − 15° C. (5° F.). Even at temperatures below its boiling-point it volatilizes so rapidly that if a drop falls on a glass plate, a portion of it freezes. The pure acid is very unstable and is rarely seen even in the chemical laboratory. The only preparation of hydrocyanic acid recognized in the United States Pharmacopeia is a 2 per cent. solution—the *Acidum Hydrocyanicum Dilutum*; this is a colorless fluid with a characteristic odor and taste, and ought not to be kept long, as it is liable to decomposition. Much of the acid used in medicine has undergone partial decomposition and is, therefore, under 2 per cent. in strength. A number of preparations recognized in the United States Pharmacopeia contain hydrocyanic acid in greater or less amounts.

Potassium cyanid (KCN, *Potassii Cyanidum*, U. S. P.) is a white, deliquescent salt, odorless when perfectly dry, but emitting the odor of hydrocyanic acid when moist. It is soluble in two parts of water. The solution has an alkaline reaction and decomposes slowly in the cold, and rapidly upon heating. Commercial preparations contain some cyanate and carbonate of the alkali. This salt is used extensively in photography, in silver electroplating, in the gilding of metals, in gold-mining, and in the manufacture of certain dyes. In some of these processes hydrocyanic acid vapors are emitted, and these may give rise to cases of poisoning. One part of hydrocyanic acid is contained in two and one-half parts of potassium cyanid; when the salt is taken into the stomach, hydrocyanic acid is set free by the action of the acid of the gastric juice, so that the effects of potassium cyanid are practically the same as those of an equivalent amount of hydrocyanic

[1] Witthaus and Becker, *Medical Jurisprudence, Forensic Medicine, and Toxicology*, vol. iv., p. 607.

acid; the salt, however, causes some corrosion at the point of application.

Physiologic Action.—When a solution of hydrocyanic acid is applied to the skin or to a mucous membrane, it causes numbness and a partial loss of sensation. Upon the central nervous system it acts first as a stimulant, causing excitement and then convulsions; the convulsions seem to be caused chiefly by the action of the poison upon the medulla and lower parts of the brain. The stage of stimulation is very brief, and is followed by paralysis of all parts of the central nervous system. The respiration is first stimulated and then paralyzed: the circulation is similarly affected. Hydrocyanic acid exercises a depressant action upon protoplasm in general: the movements and nutritive processes of both plants and animals are impaired by it. Geppert[1] showed that its effect upon protoplasm was due largely to its retarding oxidation processes; as a consequence of this action, the tissues are no longer capable of absorbing oxygen, and in the higher animals the blood, not giving up its oxygen to the tissues, retains its bright arterial color in the veins.

When hydrocyanic acid is added to drawn blood, the latter loses its power to decompose hydrogen dioxid: the blood also retains its red color much longer than does normal blood. Various combinations of hydrocyanic acid with the blood pigment have been described, but it is doubtful whether such combinations are formed in the body during life. In cases of fatal poisoning, however, the dependent parts of the body often present a bright-red color. The explanations offered for this are not very satisfactory.

Hydrocyanic acid is rapidly changed in the body: part forms sulphocyanids and is eliminated in the urine in this form. Some free acid is probably excreted by the lungs.

Hydrocyanic acid is readily absorbed from all mucous membranes. Very severe cases of poisoning have resulted from the inhalation of its vapors[2] or from its absorption from external surfaces.

Symptoms.—The symptoms of hydrocyanic acid poisoning vary according to the size of the dose and the purity of the preparation taken, and also with the condition of the stomach, whether empty or containing food. The symptoms in cases of poisoning with potassium cyanid are identical with those of hydrocyanic acid, except that the course may be slightly more prolonged. When a large quantity of the acid is swallowed, the symptoms seem to begin immediately or within a very few seconds: they are rarely delayed for more than one or two minutes. The patient may utter a cry as for help, and then fall down insensible; the respiration is at first rapid and convulsive, but soon becomes extremely slow and gasping. Convulsions are common, but in some cases the patient staggers a few steps and then falls down and dies in five minutes or less without sound or convulsion.

In most cases, however, the case is somewhat more prolonged, and

[1] Geppert, *Zeitschr. f. klin. Med.*, 1888, vol. xv., pp. 208 and 307.
[2] *Lancet*, 1899, vol. i., p. 43.

it is possible to recognize several stages. As the poison is swallowed there are an acrid taste and a feeling of constriction in the throat. Other symptoms may not begin for several seconds or even minutes, and the patient may perform a number of conscious acts, such as walking across the room, rinsing the glass from which the poison was taken, or concealing the bottle or throwing it out of the window. As a rule, however, if a fatal dose has been taken, no voluntary acts of any importance are performed;[1] the feeling of constriction in the throat is followed by salivation, nausea, and occasionally, though rarely, by vomiting; these are followed by anxiety, confusion, vertigo, and headache. There are unsteadiness of the gait and a feeling of stiffness of the lower jaw. There are palpitation of the heart and a feeling of constriction in the chest, and the respiration becomes first rapid, then slow and irregular. The inspirations are very short, the expirations greatly prolonged. The patient becomes unconscious, falls suddenly to the ground in convulsions not unlike those of epilepsy. The skin is covered with a cold sweat; the pupils are dilated and insensible to light; the eyes are glassy, staring, and very prominent, as in other cases of asphyxia. The mouth is covered with foam, which is sometimes blood-stained; the breath smells strongly of hydrocyanic acid. The pulse is at first rapid, but so weak that it can scarcely be felt. The convulsions may be general and lead to opisthotonos, or they may be confined to certain groups of muscles; thus there is often trismus. The hands are usually clenched. Involuntary evacuations of the feces and urine, also of the semen, may occur.

The convulsive stage is followed by that of depression and paralysis. The patient remains unconscious and then becomes comatose; the skin is usually cyanotic; the temperature falls; the heart is very feeble and irregular; there is complete abolition of the reflexes. The most marked symptoms, however, come from the respiration; this ceases for long intervals and then there is a short, gasping inspiration, followed by a very prolonged powerful expiration. A little later the respirations become very slow and shallow and finally cease. A few weak contractions of the heart may be noticed after the respiration has ceased. Occasionally the respiration is stertorous,[2] resembling that of apoplexy.[3] Sometimes convulsions do not occur, although the course is somewhat prolonged.[4]

In less severe cases of cyanid poisoning in which the patient finally recovers the early symptoms are as those described above; the patient falls to the ground insensible, convulsions follow, succeeded by the stage of paralysis, in which the respiration becomes slow and shallow. After a little the respiration begins to improve and the patient awakes; vomiting now frequently occurs. A feeling of constriction in the chest and weakness, causing an unsteady gait, headache, difficulty in speech, and drowsiness may continue for a few days; as a rule, however, recovery is rapid and complete. In Kolipinski's case[5] recovery was

[1] See, however, the remarkable case reported by Hickman, Case 3, below.
[2] Kelly, *Lancet*, 1879, vol. ii., p. 831.
[3] Kolipinski, *Maryland Med. Jour.*, 1898, vol. xl., p. 24.
[4] See Case 8.
[5] Kolipinski, *loc. cit.*

complete in twelve hours. In the often-quoted case of Dr. Arnold[1] the patient (Dr. Arnold) was unconscious for six hours. Before the return of full consciousness he had the most horrible sensation of impending suffocation. As soon as the first disposition to vomit was felt, consciousness was perfectly restored and there was a complete cessation of all the symptoms.

The mortality in cases of cyanid poisoning is very great; thus in a series of 455 cases the mortality was 84 per cent., and the series included many cases of slight poisoning.[2] In a series of 40 cases the mortality was 95 per cent.[3]

Cyanid poisoning may result from the inhalation of the vapors of hydrocyanic acid; the chief symptoms are a sensation of constriction of the chest, dizziness, vertigo, and insensibility. In one case there were disturbances of the vision.[4]

After poisoning with potassium cyanid, especially if the salt contains much potassium carbonate, recovery is somewhat slower, as there is much corrosion of the mucous membranes of the throat and stomach. Vomiting is more frequent in poisoning with potassium cyanid than with hydrocyanic acid.

A form of chronic poisoning occurs among gilders, photographers, and others who are engaged in the handling of hydrocyanic acid or potassium cyanid. The symptoms are headache, vertigo, paleness of the face, loss of appetite, offensive breath, and difficult respiration.

The **diagnosis** of acute cyanid poisoning is usually easy; the odor of the poison alone is often sufficient to make the diagnosis very probable. Brandy or other alcoholic beverages may, however, obscure the odor of the hydrocyanic acid.[5] If but a small dose of the cyanid has been taken, the symptoms may resemble those of nitrobenzene poisoning;[6] the symptoms in the latter case, however, are usually slow in appearing, and there is often marked cyanosis, which may continue for days.

Fatal Dose.—The smallest dose of hydrocyanic acid known to have caused the death of an adult seems to have been about $\frac{1}{2}$ of a dram (1.87 c.c.) of the 2 per cent. solution;[7] this corresponds to $\frac{6}{10}$ of a grain (0.04 gm.) of pure hydrocyanic acid. A healthy woman, aged twenty-two, died in fifteen to twenty minutes from about $\frac{9}{10}$ of a grain (0.058 gm.) contained in a lotion.[8] In another case a man took a similar dose and was insensible for four hours, but recovered. From these cases it would seem that 1 grain (0.064 gm.) of hydrocyanic acid, corresponding to 50 minims (3 c.c.) of the 2 per cent. solution, is the smallest quantity that, under ordinary circumstances, would prove fatal. Similarly, 2.4 grains (0.154 gm.) may be regarded as the minimal fatal dose of potas-

[1] Arnold, *Amer. Jour. Med. Sci.*, 1869, vol. lvii., p. 104.
[2] Witthaus and Becker, *op. cit.*, p. 611.
[3] Lesser, *Virchow's Archiv*, 1881, vol. lxxxiii., p. 198.
[4] Tatham, *Brit. Med. Jour.*, 1884, vol. i., p. 409.
[5] Howell-Thomas, *Lancet*, 1873, vol. ii., p. 522.
[6] The case of "prussic acid" poisoning reported by Cox (*Indiana Med. Jour.*, 1902, p. 156) is evidently a case of nitrobenzene poisoning.
[7] Garstang, *Lancet*, 1888, vol. ii., p. 15.
[8] Case 2.

sium cyanid. In a number of cases 5 grains (0.32 gm.) have caused death. In nearly all fatal cases of cyanid poisoning much more than the lethal dose has been taken. Recovery has also frequently followed much larger doses, but in many cases it is uncertain just how much of the acid was actually taken, as the commercial preparations vary so greatly in strength. Thus recovery has followed ½ of an ounce (15 c.c.) of the 2 per cent. solution[1] (4.8 grains—0.31 gm.—hydrocyanic acid), and even larger doses. Recovery followed the taking of what was estimated to be at least 20,[2] 40,[3] and even 50 or 60[4] grains (1.2 to 3.84 gm.) of potassium cyanid. In most of these cases, however, the exact strength of the preparation was not determined.

Fatal Period.—Hydrocyanic acid is one of the most rapidly fatal poisons known, and in many cases of poisoning the victims have been found dead within a very short time after they had been seen perfectly well. The fatal period depends upon a number of circumstances; one of the most important of these is the amount of the poison taken. As a rule, if ½ of an ounce (15 c.c.) or more of the 2 per cent. solution is taken, death occurs in from two to ten minutes. With smaller, but still fatal, doses life may be prolonged for some little time, but in most cases death occurs in less than half an hour. After 3 drams (11.1 c.c.) death occurred in twenty minutes;[5] after ½ of a dram (1.87 c.c.), in one hour and twenty minutes.[6] Probably by far the longest fatal period recorded is that in which a suicide did not die for three hours and a half after taking an unknown amount of hydrocyanic acid.[7] In the great majority of cases recovery follows if the patient lives an hour.

Death may occur in potassium cyanid poisoning in as short a time as after hydrocyanic acid;[8] but, on the other hand, a few cases are reported in which death was delayed for several—*e. g.*, two—and even twenty-four, hours.

Treatment.—In case the physician arrives before death occurs, he should wash out the stomach until the wash-water no longer has the odor of hydrocyanic acid. Some advise the addition of hydrogen peroxid or of potassium permanganate to the wash-water on the theory that these substances would convert the hydrocyanic acid into the relatively harmless oxamid, but it seems probable that this change takes place much too slowly to be of any help whatever. If the stomach-tube is not at hand, vomiting should be provoked by irritation of the pharynx or by household emetics; emetics, however, may fail entirely.[9]

Cobaltous nitrate seems to be an efficient antidote, but care must be taken not to administer an overdose, as the antidote is itself poisonous. A mixture of ferrous sulphate and magnesium oxid in water is also said

[1] Case I: Kolipinski, *loc. cit.*
[2] Higgins, *Medical Record*, 1891, vol. xl., p. 687.
[3] Ord, *Lancet*, 1886, vol. ii., p. 1174.
[4] Gillebrand, *ibid.*, 1876, vol. ii., p. 223.
[5] Kelly, *loc. cit.*
[6] Garstang, *loc. cit.*
[7] *Brit. Med. Jour.*, 1888, vol. i., p. 131.
[8] Haskins, *Boston Med. and Surg. Jour.*, 1870, vol lxxxii., p. 21. Three other cases of potassium cyanid poisoning are cited in this article.
[9] *Brit. Med. Jour.*, 1883, vol. i., p. 131; Higgins, *Medical Record*, 1891, vol. xl., p. 687.

cyanhematin that is formed by the combination of the hydrocyanic acid with the altered blood pigment (see Plate 8). This appearance is not noted, however, if the contents of the stomach were acid when the poison was swallowed.

CASES OF POISONING BY HYDROCYANIC ACID.

CASE 1.—A man, aged twenty-two, swallowed rather more than ½ of an ounce of the fresh 2 per cent. solution of hydrocyanic acid, corresponding to 4.8 grains (0.31 gm.) of the anhydrous acid. He experienced a feeling of numbness and anesthesia of the lips, quickly followed by shortness of breath and loss of consciousness. When admitted to hospital shortly afterward, pupils were widely dilated, the right a little more so than the left; pulse 86, small and compressible; respirations shallow, and ranging between 40 and 50 a minute. No cyanosis; face had a rosy tint, but the surface was cold. Rectal temperature, 97.5° F. There was soon complete insensibility, with abolition of reflexes; pulse increased to 112 and became irregular. The dyspnea was extreme, but there was no pallor of the mucous membrane or lividity, the color continuing of the same bright arterial hue throughout. There was decided trismus, rendering the introduction of the stomach-tube difficult, and for a brief space there was rigidity of the limbs. There was involuntary escape of the urine.

The treatment consisted in thorough lavage of the stomach, the hypodermic injection of whisky, camphorated ether, strychnin and atropin sulphates, the administration of whisky and ammonia by the mouth, and the application of dry heat. Sugar, albumin, casts, leukocytes, epithelial cells, and oxalate of calcium crystals were found in the urine.

There were signs of returning consciousness in two hours after admission, and recovery was rapid.

Two drops of the portion of the acid remaining in the bottle found on the patient's person caused death in two and a half minutes when placed upon the tongue of a healthy kitten.[1]

CASE 2.—A woman, aged twenty-two, swallowed an ounce of a lotion containing $\frac{9}{10}$ to 1 grain of hydrocyanic acid. The patient, who was seated in a chair, instantly jumped up, ran the distance of a few feet, and then fell to the ground insensible. There were violent convulsions, the face was distorted, and the limbs were extended and fixed in tetanic spasm. After five minutes the limbs were still extended and inflexible, the face swollen and turgid, the lower jaw spasmodically fixed, the eyelids half closed, but the eye prominent and glistening, with pupil strongly dilated. She was foaming at the mouth, breathing at long intervals with a deep, inspiratory effort, and uttering a moaning noise; pulse imperceptible at wrist. She died quietly, without any further struggle, fifteen or twenty minutes after taking the medicine. The patient had not spoken after taking the poison; she did not scream, nor was she sick; feces and urine were not passed. Postmortem four days later; limbs still rigidly extended, fingers strongly clutched, countenance turgid and distorted, jaws fixed, eyes glassy and bright. The vessels of the brain and lungs were congested; the chest evolved a strong odor of hydrocyanic acid.[2]

CASE 3.—A stout, muscular man, aged forty, drank by mistake half an ounce of a 1.48 per cent. solution of hydrocyanic acid. "The bottle was found replaced and the cupboard door put to, and it would seem that he had poured out his dose into a measure, and had drank it off standing at the cupboard; and, finding out his mistake, had put down the measure, and mechanically restoppered and replaced the bottle, closing the cupboard door, and had then run upstairs to the house surgeon, having on his way to cross the dispensary room, to open a spring door, to go up a crooked flight of fourteen stairs, across a long landing, up a second flight of eighteen stairs, and a distance of several more paces through two other doors into the house-surgeon's room." He told the house-surgeon that he had taken half an ounce of prussic acid and asked him to come to the basement; he then retraced his steps—a distance of from twenty-five to thirty paces and

[1] Shively, *Amer. Jour. Med. Sci.*, n. s., 1890, vol. c., p. 42.
[2] Letheby, *Lancet*, 1845, vol. ii., p. 99; also vol. i., p. 638.

thirty-two stairs. He stood in the middle of the room, moved his hand impatiently, and said: "Be quick—give me something." He drank some solution of ammonia and tincture of sesquichlorid of iron and tried to excite vomiting by putting his finger into his throat. After one or two abortive attempts at vomiting he fell suddenly, completely insensible. He died, without convulsions, in about ten minutes. "In a few hours after death the face regained its natural expression and color, and it was noticed that it had the hue of health, and perfectly resembled that of one asleep; but after the lapse of a few more hours it again became congested, red, puffed, and ecchymosed," and a little later face, scalp, neck, shoulders, and thighs were mottled deep red.

About 3.5 grains (0.22 gm.) of anhydrous acid had been taken.[1]

OIL OF BITTER ALMONDS.

When bitter almonds are expressed, they yield a bland, fixed oil (*Oleum Amygdalæ Expressum*, U. S. P.). When the residuary cake is submitted to distillation with water, the volatile *Oleum Amygdalæ Amaræ* (U. S. P.) is obtained. The latter is composed largely of benzaldehyd, and to a much less extent of hydrocyanic acid. These bodies are derived from the action, in the presence of water, of a ferment, emulsin, upon a glucosid, amygdalin, contained in the almonds. This action is represented by the following equation:

$$\underset{\text{Amygdalin}}{C_{20}H_{27}NO_{11}} + \underset{\text{water}}{2H_2O} = \underset{\text{dextrose}}{2C_6H_{12}O_6} + \underset{\text{benzal-dehyd}}{C_6H_5.CHO} + \underset{\text{hydro-cyanic acid}}{HCN}.$$

The benzaldehyd and hydrocyanic acid are volatile, and are, therefore, contained in the distillate. The ordinary crude oil of bitter almonds, which is official, contains from 2 to 14 per cent. of hydrocyanic acid. It was formerly much used as a flavoring agent in confectionery, etc.; at present benzaldehyd or the "synthetic oil of bitter almonds,"[2] which contains no hydrocyanic acid, is said to be largely used instead of the natural oil. The "essence of bitter almonds" is a solution of the oil in alcohol. Some pomades and salves contain oil of bitter almonds, and their use may lead to poisoning.

Of 402 cases of cyanic poisoning collected by Witthaus, 61 were due to the oil of bitter almonds; some of these were suicidal, others were accidental,[3] while in some cases the poison is alleged to have been used for criminal purposes.

The poisonous action of the oil of bitter almonds is due entirely to the hydrocyanic acid contained in it, and the symptoms are the same as those described above. In some cases, however, the effects are more slowly produced, although death may occur in ten minutes.[4] The amount of hydrocyanic acid contained in the various specimens of the oil of bitter almonds varies considerably, so that it is impossible to state with accuracy the fatal dose; it is probable, however, that 10 drops of

[1] Hickman, *Lancet*, 1866, vol. i., p. 310.

[2] The "synthetic oil of bitter almonds" must be carefully distinguished from the "artificial oil of bitter almonds"; the latter is nitrobenzene and is very poisonous. Allen (*Internat. Med. Mag.*, 1893, vol. ii., p. 126) described a case of poisoning by "oil of bitter almonds"; the intense and long-continued cyanosis and the "tar"-like character of the blood strongly suggest nitrobenzene.

[3] *Pharm. Jour. and Trans.*, 1883, 3 s., vol. xiii., pt. ii., pp. 579, 619; 1888, 3 s., vol. xiv., pt. i., p. 58.

[4] *Lancet*, 1858, vol. i., p. 128.

many preparations would be fatal. Taylor records a case in which a woman died in half an hour from 17 drops; in another case a man died in ten minutes from 2 drams (7.5 c.c.). On the other hand, with prompt treatment, patients have recovered from 6 drams (22.5 c.c.).

A child of three died in about two hours and a half after taking about 20 drops of the "essence of bitter almonds";[1] this should correspond to from 2½ to 5 minims of the essential oil, but the preparations vary much in strength.

Bitter Almonds.—Poisoning by eating bitter almonds themselves is comparatively rare, but a few fatal cases are on record.[2] Bitter almonds contain about 3 per cent. of amygdalin. Since 100 parts of amygdalin yield 5.9 per cent. of hydrocyanic acid, somewhat more than 1 ounce (31 gm.) of the almonds would be required to yield 1 grain (0.064 gm.) of anhydrous hydrocyanic acid. Bitter almonds weigh on the average about 11 grains (0.7 gm.) each; hence death may be expected to follow the eating, by an adult, of about 50 almonds. Very severe symptoms were produced in a child by eating 10 bitter almonds. In a case reported by von Maschka[3] about 2½ ounces (77.5 gm.) were taken with suicidal intent. The symptoms began in a few minutes; there were unconsciousness and convulsions in ten minutes and death in an hour and a half after the appearance of the first symptoms. Baker[4] reports a case in which a man stated that he had eaten "two handfuls" of bitter almonds; the symptoms did not appear for some time, but were then very severe. Recovery followed.

Many other plants contain amygdalin, from which hydrocyanic acid may be evolved. A few cases of severe, and even fatal, poisoning, especially in children, are reported from the eating of the kernels of the peach, the fruit of the common wild black cherry (*Prunus serotina*), the kernels of the common cherry and of the apple. The symptoms are due entirely to the hydrocyanic acid, and are those already described. The course of the poisoning, however, is usually slow. Thus a girl of five did not die for seventy hours after eating an unknown quantity of cherry-kernels. The bark and leaves also of many plants belonging to the plum family contain hydrocyanic acid in combination.

The fact that hydrocyanic acid is so widely distributed in nature has been used by the defense in criminal cases as an argument that the hydrocyanic acid found by the prosecution came from substances that had been eaten. Hence it is important to know, even approximately, the quantity of hydrocyanic acid that can be obtained from various fruits and seeds. According to Blyth, the kernels of the peach contain about 2.85 per cent. of amygdalin (= 0.17 per cent. HCN); those of the plum, 0.96 per cent. (= 0.056 per cent. HCN); those of the cherry, about 3 per cent. (= about 0.37 per cent. HCN); and apple-pips, 0.6 per cent. (= 0.035 per cent. HCN).

[1] Armstrong, *Austral. Med. Jour.*, 1880, vol. ii., p. 202.
[2] Horrocks, *Austral. Med. Gaz.*, 1898, vol. xvii., p. 252.
[3] See Blyth, *Poisons*, 1895, p. 209. [4] Baker, *Brit. Med. Jour.*, 1881, vol. ii., p. 12.

Cyanids other than Hydrogen and Potassium Cyanids.—All cyanids that are soluble in water or from which hydrocyanic acid is liberated by the action of weak acids are poisonous. Among such bodies may be mentioned the cyanids of sodium, ammonium, zinc, silver, and mercury. In cases of poisoning by mercuric cyanid the pathologic action of both mercury and hydrocyanic acid are noticed.

The double cyanids, such as potassium ferrocyanid, Prussian blue, Turnbull's blue, etc., are usually considered to be non-poisonous, but under some conditions hydrocyanic acid is split off and poisonous symptoms are produced.[1] Thus when potassium ferrocyanid is taken simultaneously with acids, fatal poisoning may occur.

Cyanic acid, the cyanates, and the sulphocyanates are nearly harmless. Cyanogen, cyanogen iodid, cyanogen chlorid, and methyl cyanid are all very poisonous.

Isolation.—There have been undoubted cases of poisoning with hydrocyanic acid where competent chemists were unable to detect the substance a few hours after its use. On the other hand, Dragendorff[2] was successful in showing the presence of the poison in the stomach of a dog four weeks after the administration of potassium cyanid and in a human corpse eight days after death, while Struve's[3] recovery of hydrocyanic acid from a mixture of potassium cyanid and organic matter that had been preserved in a closed vessel for five hundred and forty-seven days shows that a chemical examination should never be omitted where circumstances point to the use of the poison.[4] Bischoff[5] advises the analysis of the stomach, duodenum, blood, and organs rich in blood.

The separation of hydrocyanic acid from an acidified mixture of potassium cyanid and organic matter by distillation can offer no serious difficulty, but in case mercury is present, hydrocyanic acid will not be found in the distillate. On the other hand, the presence of hydrocyanic acid in the distillate may be attributed to non-poisonous potassium ferrocyanid, potassium ferricyanid, or potassium sulphocyanate,[6] all of which yield hydrocyanic acid under the conditions stated. Before proceeding with the distillation, therefore, a portion of the material is filtered or dialyzed, and the liquid is tested with an acidified mixture of ferrous sulphate and ferric chlorid. A blue color indicates either potassium ferrocyanid or potassium ferricyanid, and a red color, potassium sulphocyanate. The result of this test, taken in connection with an analysis for mercury, will determine the course of the subsequent procedure.

In the absence of mercury and compound cyanids the material under examination is thoroughly macerated with water and the mixture acidified markedly with tartaric acid, and submitted to a slow distillation

[1] See Huber, *Zeitschr. f. klin. Med.*, 1888, vol. xiv., p. 515 (poisoning by Prussian blue; recovery).
[2] *Ermittelung von Giften*, Göttingen, 1895, p. 70.
[3] *Zeitschr. f. anal. Chem.*, vol. xii., p. 14.
[4] Sokoloff, *Ber. d. d. chem. Ges.*, vol. ix., p. 1023.
[5] *Ibid.*, vol. xvi., p. 1351.
[6] Struve, *loc. cit.*

over a warm water-bath for five hours. Sokoloff's[1] contention that subsequent portions of the distillate contain more hydrocyanic acid than the first, and that the distillation should proceed for two or three days, has been shown by many chemists to be without foundation.[2] In order to facilitate the escape of hydrocyanic acid a gentle current of air[3] is passed through the material undergoing distillation and through a 2 per cent. solution of caustic potash, which is placed in the receiver for the absorption of the acid. The slight advantage of a current of carbon dioxid in preventing any oxidation of hydrocyanic acid to carbon dioxid and ammonia is more than counterbalanced by the expulsion of hydrocyanic acid from the distillate, which is sure to occur when the caustic potash contained in the receiver has become saturated with carbon dioxid.[4] The distillate will contain potassium cyanid and caustic potash, and should be used for the tests given below directly or after neutralization, according to the requirements of the test in question.

In the presence of compound cyanids the suspected material is thoroughly macerated with a solution of sodium bicarbonate, and submitted to a slow distillation. It is immaterial whether the mixture under examination is originally acid, alkaline, or neutral, since a perfectly definite condition of acidity is necessarily brought about by the presence of the bicarbonate. Upon gentle warming carbon dioxid is slowly evolved, and any potassium cyanid will be decomposed, giving off hydrocyanic acid, while the compound cyanids do not yield a trace of hydrocyanic acid under these conditions.[5] For reasons already stated it is preferable in this instance to absorb the hydrocyanic acid in a dilute solution of silver nitrate that has been faintly acidified with nitric acid.[6] The hydrocyanic acid is precipitated quantitatively as silver cyanid. The precipitate is filtered off, washed, suspended in water, and decomposed with hydrochloric acid.[7] After filtering off the silver chlorid the fluid is neutralized with caustic potash and submitted to tests that are not vitiated by the presence of potassium chlorid. In case poisoning has been accomplished with mercuric cyanid it is necessary to decompose this compound by the addition of a little sulphureted hydrogen water before the distillation is undertaken, and the employment or omission of sodium bicarbonate is determined by the presence or absence of double cyanids.[8] The use of silver nitrate in this case for the absorption of hydrocyanic acid is, of course, inadmissible. Beckwits' method[9] of removing mercuric cyanid directly from the tissues with ether offers serious experimental difficulties.

Tests.—1. A portion of the suspected fluid, which may be alkaline or neutral, is treated with a small quantity of a freshly prepared mixture

[1] *Loc. cit.*

[2] Dragendorff, *Ermittelung von Giften*, Gottingen, 1895, p. 71; Bischoff, *Ber. d. d. chem. Ges.*, vol. xvi., p. 1351.

[3] Almén, *Zeitschr. f. anal. Chem.*, vol. xi., p. 360.

[4] Struve, *loc. cit.*

[5] Autenrieth, *Arch. d. Pharm.*, vol. ccxxxi., p. 107; *Ber. d. d. chem. Ges.*, 26 Ref., p. 727; Naudin and Montholow, *ibid.*, vol. ix., p. 1433.

[6] Jacquemin, *Ann. de chim. et de phys.* (5), vol. iv., p. 139.

[7] Béchamp, *Jahresb. d. Chem.*, 1853, p. 680.

[8] Autenrieth, *loc. cit.*

[9] *Arch. d. Pharm.*, vol. xxi., p. 576.

of ferrous sulphate and ferric chlorid. Unless the material was originally alkaline, a drop or two of caustic soda is now added and the solution is warmed. Upon acidification with hydrochloric acid Prussian blue is immediately precipitated. The reaction as described will show the presence of hydrocyanic acid in a dilution of 1 : 50,000.[1]

2. A few drops of the fluid are made alkaline[2] if necessary and evaporated to dryness in a porcelain dish with a small quantity of yellow ammonium sulphid. The residue is taken up in a small quantity of very dilute hydrochloric acid; the solution is immediately filtered and treated with a trace of ferric chlorid. The red color thus produced[3] is due to the formation of ferric sulphocyanate and serves for the detection of hydrocyanic acid in a dilution of 1 : 4,000,000.[4] It is possible for this test to yield a positive result where other tests for hydrocyanic acid fail. In such a case the red color is probably attributable to the presence of sulphocyanic acid originally in the distillate, and a second test should be made without using ammonium sulphid.[5]

3. The suspected fluid is treated in turn with a dilute solution of potassium nitrite, a trace of ferric chlorid, and a sufficient amount of dilute sulphuric acid to produce a yellow color. The mixture is warmed, cooled again, and the excess of iron is precipitated with ammonia. The fluid will contain potassium nitroprussiate, which can be recognized by the violet color produced upon the addition of ammonium sulphid. Vortman claims as the limit to the sensitiveness of this reaction a dilution of 1 : 312,000.[6]

4. Ten milligrams of ferrous ammonium sulphate and 10 centigrams of uranium acetate or cobalt nitrate are dissolved in 50 c.c. of water. One cubic centimeter of the reagent is placed on a white porcelain surface, and a drop of the suspected solution, which should be neutral, is added. In case a cyanid is present, a purple color or a purple precipitate will result.[7]

5. The following test, which was first accurately described by Schönbein,[8] is usually applied to suspected organic matter before any attempt is made to isolate the poison by distillation. The material under examination is placed in a small flask and acidified with tartaric acid. The mouth of the flask is closed by a tightly fitting cork whose lower surface is provided with a slit into which is inserted a piece of filter-paper that has been wet in turn with 5 per cent. tincture of guaiacum and 0.1 per cent. copper sulphate solution. On standing for a while in a warm place the filter-paper assumes a fine deep blue color if hydrocyanic acid is present. The sensitiveness of the reaction is such[9] that one can scarcely expect to show hydrocyanic acid when this test fails, but the blue color is produced by a comparatively large number of

[1] Link and Mockel, *Zeitschr. f. anal. Chem.*, vol. xvii., p. 455; Almén, *ibid.*, vol. xi., p. 360; Carey Lea, *Amer. Jour. Sci.*, 1875, p. 122.

[2] Almén, *loc. cit.*

[3] Liebig, *Liebig's Annalen*, vol. lxi., p. 126.

[4] Link and Mockel, *loc. cit.*

[5] Struve, *loc. cit.*

[6] *Monatsh. f. Chem.*, vol. vii., p. 416.

[7] Carey Lea, *loc. cit.*; *Jahresb. der Chem.*, 1875, p. 964.

[8] *Jour. f. prak. Chem.*, vol. cvi., p. 268.

[9] Almén, *loc. cit.*; Link and Mockel, *loc. cit.*

substances,[1] among others chlorin, bromin, nitric fumes, ozone, and ammonia.

6. On treatment with silver nitrate and nitric acid soluble cyanids yield a precipitate of silver cyanid, easily soluble in ammonia or sodium thiosulphate. Under favorable conditions the precipitate will consist of clusters of radially concentric needles. When such is not the case, the substance can be distinguished from silver chlorid as follows: The precipitate is filtered off, washed with alcohol and ether, and placed in a narrow, dry test-tube with a little iodin. On gently heating, cyanogen iodid sublimes and is deposited on the cold part of the tube in beautiful pale-red crystals.[2]

FIG. 64.—Crystals of silver cyanid from very weak hydrocyanic acid; × 340.

7. To a small quantity of a 1 to 4 per cent. aqueous solution of blood is added a trace of a dilute solution of potassium ferricyanid. The color changes to brown, and the solution shows the well-known absorption spectrum of methemoglobin. Upon the addition of a drop of a neutral solution of a cyanid the color becomes bright red and the spectrum of methemoglobin disappears.[3]

Estimation.[4]—The solution in which hydrocyanic acid is to be determined is treated with 5 c.c. of strong ammonia and 2 c.c. of a 5 per cent. solution of potassium iodid.[5] Tenth normal silver nitrate solution is added from a buret a drop at a time, and with continual stirring. At the appearance of a permanent cloud in the liquid the addition of silver nitrate is discontinued.[6] One c.c. of tenth normal silver nitrate corresponds to 0.005396 gram of hydrocyanic acid—

$$AgNO_3 + KCN = AgCN + KNO_3.$$

In the gravimetric determination of hydrocyanic acid the material is distilled over borax to remove traces of chlorin, and the distillate is treated with an excess of a dilute solution of silver nitrate that contains a trace of nitric acid. The precipitated silver cyanid is collected on a weighed filter, thoroughly washed, dried at 100° C., and weighed.

[1] Struve, *loc. cit.*
[2] Blyth, *Poisons*, London, 1895, p. 205.
[3] Kobert, *Lehrbuch der Intoxikationen*, Stuttgart, 1893, p. 518.
[4] Liebig, *Liebig's Annalen*, vol. lxxvii., p. 102.
[5] Denigès, *Ann. de chim. et de phys.* [7], vol. vi., p. 381; Shurwood, *Jour. Amer. Chem. Soc.*, vol. xix., p. 400.
[6] See also Gregory, *Zeitschr. f. anal. Chem.*, vol. xxxiii., p. 30.

BENZENE.

Benzene[1] (benzol, C_6H_6) is a colorless, very inflammable liquid, boiling at 80.5° C. (176.9° F.), which is obtained from coal-tar by distillation. When benzene is taken internally, or when its vapors are inhaled, a condition of narcosis is produced; at the same time there is a stimulation of parts of the central nervous system, leading to muscular tremor and convulsions.

A number of deaths from benzene have been reported: some of these have resulted from the poison being swallowed by suicides, but most of them have been due to the accidental inhalation of the vapors in factories. The chief symptoms are headache, dizziness, flushing, then cyanosis of the face, hallucinations, and delirium, followed in many cases by convulsions and coma. In one fatal case a woman died in twelve hours after swallowing about 1 ounce (30 c.c.) of nearly pure benzene.[2]

Santesson[3] has described a form of chronic poisoning occurring in factories in which benzene was extensively employed as a solvent for rubber. The chief symptoms resulted from numerous small hemorrhages caused by fatty degeneration of the arteries.[4]

Detection.—The separation of benzene from the tissues, though somewhat tedious, can be accomplished without serious difficulty by a process quite similar to that which serves for its isolation from coal-tar. The material under examination is submitted to successive fractional distillations, the distillates being shaken out alternately with acid and alkali, and the fraction of the distillate selected that passes over between 60° and 95° C.

Benzene serves as the starting-point in the preparation of a countless number of characteristic aromatic compounds, of which perhaps the easiest to prepare and identify is anilin. The oil is treated with three or four times its weight of fuming nitric acid in a flask under an inverted condenser, and enough heat is applied to start the reaction. The product is poured into cold water, and the oil-drops of nitrobenzene that fall to the bottom may be converted into anilin by the method given under Nitrobenzene.

NAPHTALIN.

Naphtalin is a hydrocarbon obtained from coal-tar. It is used often in the form of "moth-balls" or under the name of "tar camphor," as a poison to insects. It has been used to a limited extent in medicine as an external application in parasitic skin diseases or to wounds, and internally as an intestinal antiseptic and vermicide.

Properties.—Naphtalin ($C_{10}H_8$, *Naphtalinum*, U. S. P., naphtalene)

[1] This substance must not be confounded with benzin; the latter is a commercial product obtained in refining ordinary American petroleum.

[2] Kelynack, *Gaz. méd. de Paris*, 1893, p. 541; also *Med. Chronicle*, 1893–94, vol. xix., p. 112.

[3] Santesson, *Arch. f. Hygiene*, 1897, vol. xxxi., p. 336.

[4] Santesson, *Skand. Arch. f. Physiol.*, vol. x., p. 1.

is a white, crystalline mass with a coal-tar odor and a hot aromatic taste. It is soluble in alcohol, chloroform, ether, and the oils, but insoluble in water. It melts at 80° C. (176° F.) and boils at 217.2° C. (423° F.). It is slowly volatilized on exposure to the air.

Although naphtalin is classed with the antiseptics of the carbolic acid series, animals poisoned with it do not exhibit the ordinary symptoms of poisoning by members of this series, but suffer from diarrhea and rapid loss of flesh. There are also symptoms of parenchymatous nephritis. One of the most interesting effects of naphtalin upon the lower animals is the production of cataract. Other changes in the eye, such as the appearance of small bright spots (crystals) in the vitreous humor and retina, and degenerative changes in the retina and optic nerve, and subretinal effusions also occur.[1]

Symptoms.—In mild cases of poisoning resulting from the inhalation of powdered or vaporized naphtalin the symptoms were headache, malaise, and vomiting. Applied to wounds, it causes burning pain.

Large doses—2 or 3 drams (7.8 or 11.7 gm.) daily, for example—taken by the stomach cause vomiting; smaller doses, chiefly colicky pains, diarrhea, and tenesmus.

Important symptoms arise from the urinary organs: a burning sensation in the urethra, pain in the bladder and kidney region, and, more rarely, strangury. The urine is dark brown or becomes dark on standing: it contains α- and β-naphtol, and sometimes albumin. In one case acute nephritis and death occurred in five days after the use of naphtalin.[2]

In one case 8 grains (0.52 gm.) caused excessive vomiting and purging and great abdominal pain, followed by nephritis.[3]

A few cases of injury to the cornea are reported among workmen exposed to the vapors of nitronaphtalin.[4]

Treatment.—The symptoms have usually disappeared on the withdrawal of the drug.

Tests and Detection.—Naphtalin may be isolated by distillation with steam and extracting the distillate with ether. The ethereal solution forms with picric acid a yellow crystalline compound; 0.1 gram of dry naphtalin dissolves without color in 2.5 grams of melted chloral hydrate, and remains colorless after ten minutes' warming on the water-bath (α-naphtol becomes red, β-naphtol blue). If the above mixture be warmed with five drops of hydrochloric acid, a pale rose color may develop (α-naphtol becomes dark-greenish blue, β-naphtol yellow); if zinc be added to the above mixture, a violet or brown color develops (α-naphtol becomes blue violet, β-naphtol dark brown, and both become fluorescent with alcohol).

[1] Saemisch, *Handbuch der ges. Augenheilk.*, vol. xi., pt. ii., p. 128.

[2] Lewin, *Nebenwirkungen der Arzneimittel* 1899, p. 541.

[3] Otte, *Chinese Med. Missionary Jour.*, 1897, vol. xi., cited in **U. S. Disp.**, eighteenth ed., p. 893.

[4] See Silex, *Zeitschr. f. Augenheilk.*, 1901, vol. v., p. 178.

OILS OF TURPENTINE, SAVIN, AND CEDAR.

A number of plants belonging to the Coniferæ contain volatile oils that are of some interest in toxicology; the most important of these are the oils of turpentine, savin, and cedar.

OIL OF TURPENTINE.

Oil of turpentine (*Oleum Terebinthinæ*, U. S. P.) is a volatile oil distilled from turpentine, the latter (*Terebinthina*, U. S. P.) being an oleoresin obtained from *Pinus palustris* and other species of Pinus. When pure, oil of turpentine is composed of one or more terpenes, but, as usually seen, it contains oxidation products of these hydrocarbons. It is a light yellowish, highly inflammable oil, moderately soluble in alcohol, and very slightly so in water. It has a hot, burning taste, and when applied to the skin, causes irritation and redness. It is extensively used in the arts as a solvent.

Poisoning with oil of turpentine has resulted from its use in medicine, as, *e. g.*, an anthelmintic, or has been due to accident or occasionally to the use of the substance for the purpose of suicide or for securing abortion. The vapors are also poisonous.

When a large dose is taken internally, there is a sensation of warmth in the throat and stomach, followed by abdominal pain, vomiting, and diarrhea. The pulse is weak, and the respiration becomes slow and irregular; then follow, in some cases, nervous symptoms—excitement, muscular incoördination, and convulsions—and death in coma. There is usually great irritation of the urinary tract, as shown by strangury, hematuria, albuminuria, and often painful erections. The urine has the odor of violets. Sometimes there is complete suppression of the urine. Various forms of skin eruptions have been described.

Severe poisoning may result from the long-continued inhalation of the vapors of turpentine, as by painters or varnishers or by those who sleep in freshly varnished rooms. The symptoms are headache, dizziness, bronchitis, and often irritation of the kidneys.[1] Tolerance is readily established.

The **lethal dose** of the oil of turpentine is not known. An infant fourteen weeks old died in fifteen hours after taking half an ounce (15 c.c.) of the drug.[2] In another case an infant, aged five months, died soon after taking a teaspoonful. On the other hand, an infant of fourteen months recovered after taking 4 ounces (120 c.c.). A woman died in a few hours from about 6 ounces (180 c.c.).[3]

The **treatment** consists in the evacuation of the stomach and the administration of stimulants. Little is known about the **postmortem appearances.**

OIL OF SAVIN.

Savin (*Sabina*, U. S. P.) is the tops of *Juniperus Sabina*, an evergreen growing in Europe and said to occur also in the northern part of

[1] Reinhard, *Deutsch. med. Wochenschr.*, 1887, vol. xiii., p. 256.
[2] Miall, *Lancet*, 1869, vol. i., p. 360.
[3] Maund, *Amer. Jour. Med. Sci.*, n. s., 1858, vol. xxxvi., p. 561.

the United States. The active principle is contained in an oil, *Oleum Sabinæ* (U. S. P.), which is closely related to the oil of turpentine; like the latter it is a powerful local irritant.

The tops of savin, in the form of a decoction, have been used by the laity from the most ancient times as an abortifacient; more recently the oil has been used for a similar purpose. Most cases of death from savin have resulted from such a practice.

The **symptoms** of savin poisoning are violent abdominal pains, bloody vomiting and purging, diminution of the urine, which may be

FIG. 65.—Savin (*Juniperus sabina*).

bloody, strangury or anuria, irregular respiration, unconsciousness, convulsions, and death in collapse. Abortion frequently occurs in pregnant women,[1] but it is doubtful whether savin has any specific action upon the uterus, although it is used in treating amenorrhea; the abortion may be the indirect result of the hyperemia of the pelvic organs caused by the irritation of the intestines. In a number of cases death has occurred without expulsion of the fetus.

[1] Santesson (*Skand. Arch. f. Physiol.*, 1901, vol. xi., p. 228) describes a case in which a woman took small doses of savin for a period of fifty days. A month after the last dose a five months' fetus, said to be macerated, was expelled. The question came up in court whether the abortion could be attributed to the savin; the evidence seems to be against such a view (see Lewin and Brennig, *Die Fruchtabtreibung durch Gifte*, Berlin, 1899).

Fatal Dose and Fatal Period.—As the victims usually gather the tops of the plants themselves and make an infusion, of which they drink more or less, it has not been possible to determine how much savin is required to produce death; moreover, in most cases much of the oil is volatilized by the boiling. The medicinal dose of the oil for non-pregnant women is from 5 to 10 drops. A medical man in England who gave a pregnant woman 14 drops of the oil daily divided into three doses was convicted of administering the drug with intent to procure miscarriage.[1]

Death may occur in twelve hours or it may be delayed for two or three days. In a case described by Blyth[2] a woman died in about twenty-six hours from an unknown quantity of infusion of savin tops. Postmortem the pharynx was found reddened and the gullet congested: the stomach was congested and contained savin tops. The odor of savin is usually noticed in the stomach and sometimes in other organs.

The **treatment** consists in the evacuation of the stomach and the administration of demulcent drinks; stimulants may be necessary in the stage of collapse.

OIL OF CEDAR.

The tops of *Juniperus virginiana*, the red cedar, a tree growing in all parts of the United States, are often substituted for those of *Juniperus sabina*, from which they are distinguishable with difficulty. Red cedar contains a volatile oil that is very closely similar to that of savin; it has been used as an abortifacient and for suicidal purposes with fatal results.[3] S. C. Wait[4] reported 4 cases of poisoning by cedar oil. In 3 of these cases the poison was taken for the purpose of securing abortion; in none of the cases was the fetus expelled, while in 2 of them the woman died. In the fourth case the poison was taken with suicidal intent, the patient recovering. The symptoms in these cases were burning in the stomach, vomiting, convulsions, coma, and a slow pulse. The vomited matter smelt strongly of cedar oil. After death evidences of gastro-intestinal irritation were found. In the 2 fatal cases death occurred in about an hour after the poison was supposed to have been taken. One of the patients who recovered had taken a large teaspoonful, the other an ounce, of the oil. The treatment consisted in the administration of emetics. The doses in the fatal cases are not stated.

In a case reported by Brown[5] a pregnant woman recovered after taking ½ an ounce (15 c.c.) of the oil of cedar; bloody matter was vomited and there were convulsions, followed by stupor. There was suppression of the urine for thirty-six hours. Abortion did not occur. An entirely similar case is reported by Holley.

Bolles reports a case in which a woman died twenty-six hours after

[1] *Med. Times and Gaz.*, 1852, vol. i., p. 404. [2] Blyth, *Poisons*, 1895, p. 459.
[3] Ellyson, *Therap. Gaz.*, 1890, vol. vi., p. 371.
[4] Wait, *Boston Med. and Surg. Jour.*, 1849, vol. xl., p. 469.
[5] Brown, *Med. News*, 1893, vol. lxiii., p. 15.

liquid acid proved fatal in twenty-three hours to a girl of seventeen.[1] It is said that most cases in which 60 grains (3.9 gm.) of the acid were taken and retained and the proper treatment was not given ended fatally. Six or 7 grains (0.44 gm.) have caused severe symptoms. On the other hand, recovery has frequently followed the taking of 1 ounce (30 c.c.). In one case abortion occurred after $1\frac{1}{2}$ ounces (45 c.c.), but the girl recovered.[2] In another case a patient recovered, under very prompt treatment, from "nearly an ounce and a half."[3] The fatal dose depends largely upon the promptness with which treatment is given.

Very small quantities of phenol when introduced into wounds or into the body cavities will cause death. Thus in one case a uterine douche containing 1 dram (3.9 gm.) of carbolic acid to a quart of water caused the death of a woman in one hour and forty minutes, although much of the liquid was returned; the symptoms were thirst, vomiting, rise of temperature to 108.5° F., and violent delirium.[4] From 15 to 30 grains (1 to 3 gm.) may be regarded as a fatal dose under such circumstances. A little more than 3 grains (0.2 gm.) injected into the rectum of a child aged eight caused severe poisoning.[5]

Fatal Period.—The course of carbolic acid poisoning is rapid. In a large percentage of cases death occurs within one or two hours and it is not often delayed more than twelve hours. After very large doses death may occur within a few minutes. In one case a girl of seventeen died within ten minutes after taking about 1 ounce (30 c.c.).[6] In another case about a quart of the crude acid was swallowed, but death did not take place for seven hours. In exceptional cases death may be delayed for sixty hours, or even for four or five days. Death may result after several—*e. g.*, six—days from pneumonia caused by the aspiration of vomited matters into the lungs during unconsciousness.[7]

Death may occur within a very few minutes after the external application of carbolic acid, or it may be delayed for several days.

Treatment.—If the poison has been swallowed, the stomach should be washed out with warm water; evacuation of the stomach by emetics is much less satisfactory. In some cases emetics fail entirely.[8] Lime suspended in syrup has been highly recommended in the hope that an insoluble compound may be formed in the stomach. Sodium sulphate, which has also been recommended, seems to be of little use except in so far as it acts as a purgative. Great benefit seems to have followed the administration of milk or white of eggs; these act not only as demulcents, but the albumin combines with the phenol to form compounds that do not dissolve easily. In cases of coma warm baths with cold affusions do much good. Cardiac and respiratory stimulants are also indicated. Sometimes artificial respiration is necessary.

[1] Wilkinson, *Therap. Gaz.*, 3 s., 1892, vol. viii., p. 220.
[2] Taliaferro, *Weekly Med. Rev.*, 1883, vol. viii., p. 55.
[3] Cassidy, *Dominion Med. Monthly*, 1896, vol. vi., p. 488.
[4] Edmunds, *Med. and Surg. Reporter*, 1887, vol. lvii., p. 345.
[5] *Med. Rec.*, vol. xxviii., p. 601.
[6] Marwood, *Austral. Med. Gaz.*, 1893, vol. xii., p. 78.
[7] Schleicher, *Deutsch. med. Wochenschr.*, 1891, vol. xvii., p. 9.
[8] See Case 2.

STOMACH AND UPPER PORTION OF SMALL INTESTINE AFTER POISONING WITH CARBOLIC ACID (VON HOFMANN).

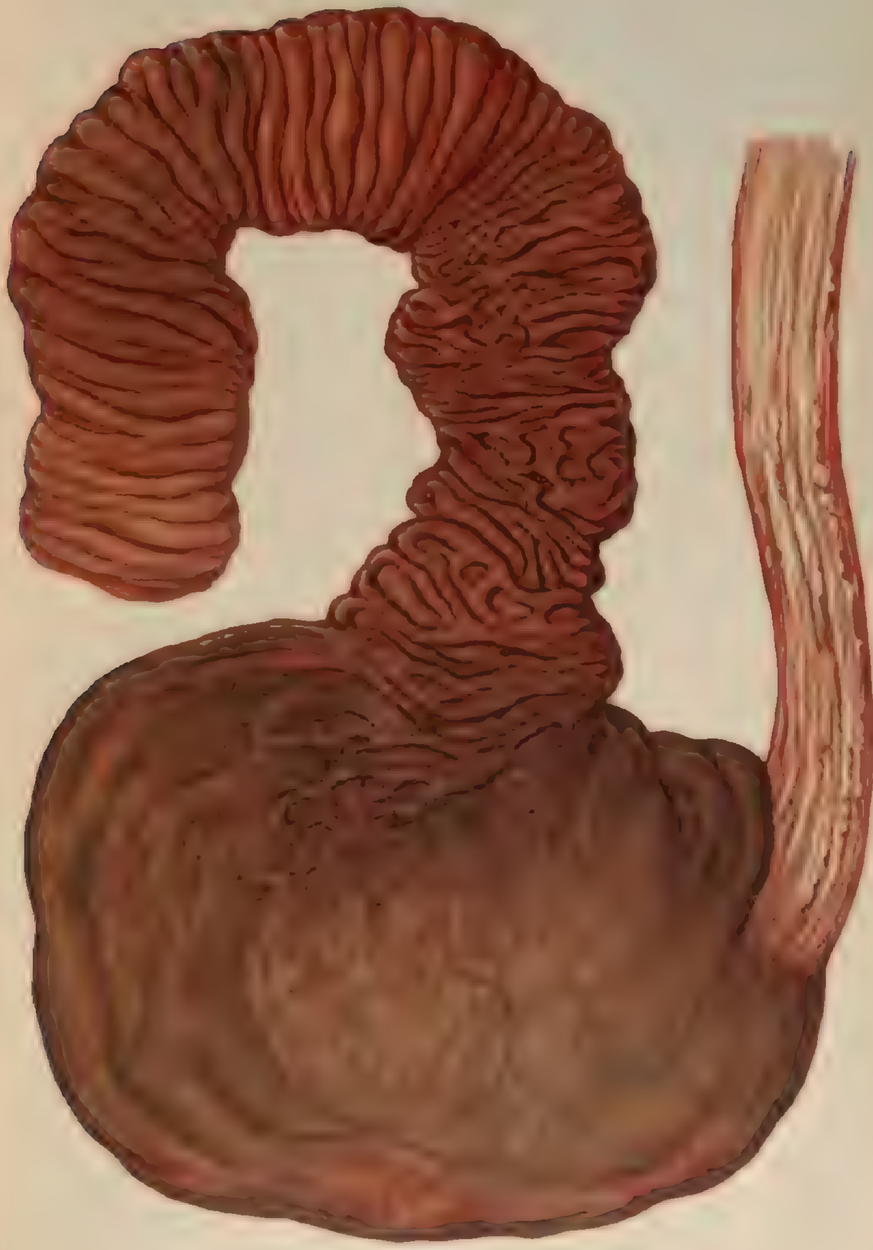
A man of fifty-one years took about 80 c.c. (between 2 and 3 fluidounces) of 90 per cent. carbolic acid. He vomited immediately, became unconscious, and died at the end of about half an hour.

At the postmortem examination the lips, mouth, and pharynx were found corroded and of a grayish-white color. The epithelium of the esophagus was arranged in white longitudinal folds, through which the mucous membrane showed pale red in color.

The stomach was much distended and contained about 500 c.c. (about 17 fluidounces) of a whey-like fluid having a distinct odor of carbolic acid. The mucous membrane of the stomach was pale red and presented prominent, well-filled vessels. The fundus of the stomach showed no marked alteration, but in the region of the pylorus, as also along the lesser curvature, the mucous membrane was arranged in rigid folds, thickened, of a grayish-red color, with milky white, cloudy epithelium. This cauterization, giving an appearance as though the tissue were cooked, penetrated in certain places to the peritoneum, and it also affected the superficial portion of the adjoining surface of the liver. It increased in intensity toward the pylorus, and continued uniformly through the entire duodenum to the beginning of the jejunum, where the mucous membrane again became soft and relaxed.

The slight cauterization of the stomach as a whole is accounted for by the fact that it was well filled with fluid at the time the poison was taken.

PLATE 9.



Much has been said recently about the use of alcohol as an "antidote" to carbolic acid. Dr. Phelps[1] stated in 1899 that Dr. Powell had observed that the escharotic action of carbolic acid in wounds, on the hands, and on mucous membranes could be prevented by the use of alcohol. It has also been shown that the danger of absorption of carbolic acid from wounds and other surfaces is much less if the acid is washed off with alcohol.[2] According to Honsell,[3] the explanation of this action is that the carbolic acid is very soluble in alcohol, and so much of it is removed. These observations have led to the use of alcohol as a remedy in cases in which carbolic acid has been taken internally; some extraordinary results have been claimed for this method of treatment. From the present state of our knowledge it seems very probable that the best method of treating a case of carbolic acid poisoning would be to administer alcohol, provided the stomach could be washed out immediately afterward.[4] To administer a large dose of alcohol and to leave it in the stomach, as has been done, would seem to be an extremely dangerous practice. It is true a number of cases have been reported in which this was done and the patients have recovered, but there is no reason to suppose that the alcohol acts in any sense as an "antidote."[5]

Postmortem Appearances.—When the poison has been taken by the stomach, there may be brownish, shrunken patches on the skin about the mouth. The mucous membrane of the mouth, esophagus, and stomach may be white, corrugated, and partly detached, and the edges of the affected parts are often hyperemic; if the case is one of long duration, these patches are red. In many cases the mucous membrane of the stomach and intestines is reddened and inflamed (see Plates 9 and 10). The urine is usually of a dark or dark-greenish color. The odor of the poison may be noticed in the body and in the urine. The blood is usually dark and fluid; the brain, the meninges, the lungs, the liver, and the spleen are often congested.

Cases of Carbolic Acid Poisoning.

Case 1.—Woman, aged sixty, took 2 ounces (60 c.c.) of pure carbolic acid. Was found, shortly afterward, unconscious; pulse thready and scarcely perceptible; extreme dyspnea; lips cyanotic; conjunctival and pupillary reflexes absent. Rectal temperature, 96° F. A stomach-tube was introduced into the pharynx and 4 ounces (120 c.c.) of alcohol were poured in. After two or three minutes the tube was introduced into the stomach and the organ washed out with warm water; the washing was repeated with diluted alcohol. Signs of improvement quickly appeared. Strychnin and other cardiac stimulants were administered. In an hour and a half the patient was cheerful and felt little discomfort. The urine, which had to be drawn off with a catheter, was brownish black, but contained no albumin. Recovery was complete in three days.[6]

Case 2.—Adult male took 1 ounce (30 c.c.) of pure carbolic acid. Patient seen

[1] *New York Med. Jour.*, 1899, vol. lxix., p. 62; see also Frazer, *Medical Record*, 1895, vol. xlviii., p. 741.

[2] Adams, *New York Med. Jour.*, vol. lxx., p. 780.

[3] *Beit. z. klin. Chir.*, 1901, vol. xxx., p. 328.

[4] See Case 1.

[5] . . e Wallace, *N. Y. Univ. Bull. Med. Sci.*, vol. ii., p. 58.

[6] . . dman, *Medical Record*, 1900, vol. lviii., p. 70.

in a few minutes. There was a strong odor of phenol on breath; the lips were stained white. One-tenth of a grain (0.0065 gm.) of apomorphin injected. No emesis having occurred, the dose was repeated in five minutes, but vomiting did not take place. Patient soon had difficulty in swallowing; the face, which was at first flushed, soon became pale and slightly cyanotic. Within a few, perhaps seven or eight, minutes after the poison was taken there were tonic spasms of the voluntary muscles with muttering delirium; patient seemed unconscious of his surroundings. A quart of milk was pumped into the stomach. Pallor increased and the body became cold to the touch. Respiration labored, pulse weak and intermittent, pupils contracted. Patient did not at any time complain of pain. He was placed in bed and various stimulants were given. Respiration ceased about fifty minutes after the poison was taken. Heart was found to be beating feebly. Artificial respiration was resorted to. In about a minute patient began to breathe again and continued to do so for thirty minutes, when both respiration and heart ceased. No autopsy.[1]

Isolation.—In most cases of poisoning by carbolic acid the odor is distinctly noticeable. The tendency of the poison to combine with sulphuric acid is such that the sulphates present in the system may be completely used up in the formation of phenol sulphuric acid, and the urine under such conditions will fail to yield a precipitate when treated with barium chlorid.

For the isolation of the poison, the material under examination is thoroughly macerated with 5 per cent. sulphuric acid and submitted to distillation with steam (see Fig. 24, p. 321) until the liquid that passes over fails to respond to Millon's test. As carbolic acid is not appreciably soluble in petrolic ether, the distillate may be shaken with this solvent for the removal of various impurities[2] and the carbolic acid finally extracted with ether or benzene. As salicylic acid responds to many of the tests for carbolic acid, it may be well to exclude this substance by making the distillate alkaline in the cold with sodium carbonate and extracting repeatedly with ether. Under these conditions carbolic acid is taken up by the ether, while sodium salicylate remains in the aqueous fluid.

It should be noted that combined carbolic acid is a normal constituent of the body, and that its amount is materially increased under certain pathologic conditions. It is, therefore, always necessary to supplement the extremely sensitive[3] tests given below by a quantitative determination. This is best accomplished by the method of Kossler and Penny, but as the limits of this book do not admit an adequate description of the method, the reader is referred to the original article[4] and to an excellent abstract by Huppert.[5]

Although the analyst may reasonably expect to find the greater part of carbolic acid in the combined form, it may, nevertheless, happen, and especially after the ingestion of large quantities, that the presence of the poison in the free condition may be demonstrated. It is, therefore, advisable to make a preliminary distillation of the material after faintly acidifying with acetic acid. Any carbolic acid that can be shown in the

[1] King, *Medical Record*, 1897, vol. li., p. 158.
[2] Jacobsen, *Zeitschr. f. anal. Chem.*, vol. xxv., p. 607.
[3] Almén, *ibid.*, vol. xvii., p. 170. [4] Kossler and Penny, *ibid.*, vol. xvii., p. 117.
[5] Neubauer and Vogel, *Analyse des Harns*, Wiesbaden, 1898, p. 785.

PLATE 10.

distillate under these conditions must have been present in the uncombined state[1] and will have greater toxicologic significance than that obtained by the distillation of a fluid that is highly acid with sulphuric acid.

Tests.—1. Solutions of carbolic acid are colored red when boiled with Millon's reagent.[2] The test serves to detect 1 part of carbolic acid in 2,000,000 parts of water, but the red color is produced also by all monophenols and by proteids.

2. Solutions of carbolic acid are colored intensely bluish-violet by a trace of ferric chlorid. The presence of mineral acid, ammonia, or an excess of the reagent is prejudicial to the production of the blue color, and the reaction fails entirely in the presence of alcohol.

3. On warming carbolic acid in substance with caustic potash and chloroform a product is formed which dissolves in alcohol with a carmin-red color. The color is removed by acids and restored by alkalis.[3] The test is given equally well by resorcin.[4]

4. To a dilute aqueous solution of carbolic acid that contains a trace of an alcoholic solution of ethyl nitrite is carefully added some concentrated sulphuric acid in such a manner that the liquids do not mix. A rose-colored ring will form at the surface of contact. The reaction will show the presence of 1 part of carbolic acid in 2,000,000 parts of water.[5]

5. From extremely dilute solutions of carbolic acid, bromin water precipitates white crystalline tribromphenol.[6]

6. On warming a solution of carbolic acid with one-fourth of its volume of ammonia and a few drops of calcium hypochlorite solution, a transient blue color is produced that returns upon the addition of more hypochlorite or by shaking with air. Instead of hypochlorite solution, vapors of bromin may be employed.[7]

7. To 50 c.c. of water add 3 drops of anilin and dilute from 5 to 10 drops of the mixture with 10 c.c. of water. Add as much sodium hypochlorite solution as will serve to change the blue color that is first formed into a brown, allow to stand a few minutes, and add the solution supposed to contain phenol and previously treated with a few drops of ammonia. In the presence of phenol a permanent blue color will be produced. This is Dragendorff's modification[8] of Jacquemin's well-known reaction.[9]

Jacobsen[10] claims that the greater part of the poison will be found in the blood and the liver, and Jacquemin[11] proposes the following short

[1] Reale, *Maly's Jahresbericht d. Thierchemie*, 1891, p. 404.

[2] Millon, *Comptes rendus*, vol. xxviii., p. 40. Dissolve mercury in an equal weight of 63 per cent. nitric acid, warming gently to start the reaction, and dilute the product with twice its volume of water. After standing overnight decant the clear reagent.

[3] Guareschi, *Ber. d. d. chem. Ges.*, vol. v., p. 1055.

[4] Lustgarten, *Monatsh. f. Chem.*, vol. iii., p. 719.

[5] Eijkman, *Zeitschr. f. anal. Chem.*, vol. xxii., p. 576.

[6] Landolt, *Ber. d. d. chem. Ges.*, vol. iv., p. 770; Benedickt, *ibid.*, vol. xii., p. 1005.

[7] Cotton, *Bull. de la Soc. Chim.* (2), vol. xxi., p. 8.

[8] *Ermittelung von Giften*, Gottingen, 1895, p. 121.

[9] *Zeitschr. f. anal. Chem.*, vol. xv., p. 367.

[10] *Loc. cit.*

[11] *Loc. cit.*

method of preparing an alcoholic extract to which his test may be applied. One hundred grams of blood or finely cut liver are digested for an hour with 2 per cent. sulphuric acid; the liquid is strained through a cloth, treated with an equal volume of 30 per cent. alcohol, and filtered. Thirty c.c. of the clear fluid are used for the test given above.

The **dihydroxybenzenes** (pyrocatechin, resorcin, and hydroquinon) have been used to a limited extent in medicine and have caused a few cases of poisoning. The symptoms are very similar to those of carbolic acid poisoning. The stimulating effect of these bodies upon the nervous system, however, is more marked than with phenol, for convulsions have occurred in some cases.

In one case 2 drams (7.76 gm.) of resorcin, taken internally, caused cold sweats, stupor deepening very rapidly into collapse, with complete abolition of reflex movement. The temperature fell to 94° F.; the urine was of an olive-green color. Recovery was rapid.[1]

Guaiacol (methyl-pyrocatechin) has caused a few deaths.[2] Most cases of poisoning have resulted from its extensive use, both internally and externally, in the treatment of tuberculosis. The symptoms are irritation of the alimentary tract, loss of consciousness, fall of temperature, and collapse. The urine shows the same changes as after carbolic acid. In one case death occurred in a tuberculous patient eighteen hours after the external application of 30 grains (1.95 gm.).[3] A number of preparations containing guaiacol, or derivatives of guaiacol, have been used in medicine; their effects are similar to those of guaiacol.[4]

Creasote consists largely of guaiacol and creosol. Its effects upon man are very similar to those caused by guaiacol. It is used extensively as an application in toothache, and its easy accessibility has led to a few cases of accidental as well as to criminal poisoning.[5] About 2 drams (7.4 c.c.) caused the death of an adult woman in thirty-six hours, while another patient recovered after drinking 6 drams (22.2 c.c.).[6]

Pyrogallol (pyrogallic acid, trihydroxybenzene, $C_6H_3(OH)_3$) is used, in the form of an ointment, in the treatment of skin diseases, especially of psoriasis. It is readily absorbed by the skin, especially from the diseased skin, and has caused a few cases of poisoning in this manner.[7] It has also been used for suicidal purposes, and solutions intended for photographic purposes have been mistaken for alcoholic liquors, with fatal results.

The chief **symptoms** in poisoning by pyrogallol result from the effect of the poison upon the red blood-corpuscles; the latter become

[1] Murrell, *Med. Times and Gaz.*, 1881, vol. ii., p. 486.

[2] Wyss, *Deutsch. med. Wochenschr.*, 1894, vol. xx., pp. 296, 321; this paper is abstracted by Friedenwald, *Maryland Med. Jour.*, vol. xxxi., p. 71.

[3] Lewin, *Lehrb. der Toxikol.*, 1897, p. 216.

[4] Mosetig-Moorhof, *Deutsch. med. Wochenschr.*, 1894, vol. xx., p. 168.

[5] Manouvriez, *Ann. d'hyg.*, 3 s., 1882, vol. vii., p. 175; Purckhauer, *Friedreich's Blätter*, Nuremburg, 1883, vol. xxxiv., p. 430 (murder of infants by creasote).

[6] Schulze, *Münch. med. Wochenschr.*, 1894, p. 219; see Freudenthal, *Medical Record*, 1892, vol. xli., p. 456.

[7] Neisser, *Zeitschr. f. klin. Med.*, 1880, vol. i., p. 88.

shrunken and lose their hemoglobin. The hemoglobin is converted into methemoglobin, and the blood, as a consequence, assumes a reddish-brown color. Icterus may follow, and the decomposition-products of the red corpuscles cause nephritis with the appearance of albumin, epithelial cells, and blood in the urine; or the nephritis may lead to uremia. These blood changes lead to headache, cyanosis, chills, vomiting, diarrhea, and strangury; the pulse becomes small, and the urine assumes a dark-brown color from the presence of methemoglobin, hematin, etc. In fatal cases the cyanosis becomes intense, tremors develop, and death occurs in a state of collapse.

The **fatal dose** is not known, but 75 grains (4.88 gm.) contained in about 3½ ounces (108.8 gm.) of salve are said to have been fatal. Banerji[1] reports a case in which two patients stated that they had each taken much more than 1 dram of pyrogallic acid internally; the symptoms were not very severe, and recovery soon followed. Smith[2] reports a case in which a man recovered after taking 120 grains (7.8 gm.) of the poison dissolved in alcohol, while another man who drank a quantity of the same liquid containing about 250 grains (16.25 gm.) died in five days from symptoms of nephritis. In another case the patient died in a state of coma in two or three days after taking 225 grains (14.6 gm.).[3]

The **treatment** consists in removing the poison from the skin, or, if it has been taken internally, in washing out the stomach. Stimulants and bleeding, followed by saline infusions, are recommended. The inhalation of oxygen is said to have been beneficial.

Postmortem[4] the skin has been found colored dark at the point of application. The kidneys were enlarged and were almost black; the pelvis of the kidney, the ureters, and the bladder contained a bloody liquid. Icterus has also been noticed.

Detection.—Pyrogallol may be extracted with ether as in an ordinary procedure for alkaloids. It reduces salts of silver and mercury to the metals, changes ferric to ferrous salts, is colored violet by dilute caustic potash, and produces a red color with Millon's reagent. In cases of pyrogallol poisoning the urine sometimes emits the odor of trimethylamin when treated with potassium hydroxid.

NAPHTOL.

Two naphtols, the alpha- and beta-compounds, may be formed from naphtalin. They bear the same relation to naphtalin that carbolic acid does to benzene. The beta-compound has been used in medicine as an external application in various skin diseases, especially in scabies, and internally as an intestinal disinfectant; it is occasionally used as a food preservative.

Properties.—β-naphtol (*Naphtol*, U. S. P.) is a white or yellowish-white, crystalline powder with a faint phenol odor and a hot taste. It is soluble in 1000 parts of cold water, readily soluble in ether, alco-

[1] Banerji, *Lancet*, 1892, vol. ii., p. 308.
[2] Smith, *Pacific Med. Jour.*, 1891, vol. xxxiv., p. 456.
[3] Dalché, *La Semaine méd.*, 1896, vol. xvi., p. 211.
[4] Neisser, *loc. cit.*

SALICYLIC ACID.

Salicylic acid (orthohydroxybenzoic acid, $C_6H_4.OH.COOH$) occurs in the form of small, white, needle-like crystals or as a light crystalline powder; it is slightly soluble in cold water, and very soluble in alcohol and ether. It has a sweetish, afterward an acrid, burning taste.

Most of the cases of poisoning with salicylic acid have been medicinal and have resulted from the extensive use of the substance and its salts in the treatment of rheumatism. It is often added to articles of food and drink (jellies, milk, beer, meats, catsups, sauces, etc.) as a preservative,[1] and has caused toxic symptoms when taken in this way.

The **symptoms** in a mild case of poisoning, such as is sometimes seen in the treatment of acute rheumatism, are a feeling of fulness in the head, with roaring sounds in the ears, dimness of vision, profuse perspiration, confusion, and dulness. With large doses of the free acid there is intense irritation of the throat and stomach, leading to vomiting and difficulty in swallowing; later there may be diarrhea. Flushing of the face, distressing dyspnea, weak pulse, subnormal temperature, and, in more severe cases, convulsions, coma, and perhaps death in a state of collapse occur. Delirium and hallucinations have been described. In alcoholic patients these symptoms may not be unlike those of delirium tremens. The urine sometimes contains blood pigments. Eczema and other skin eruptions may appear, and dimness of vision and deafness may continue for some time.

Long-continued use of salicylic acid and its salts has led to a form of chronic poisoning[2] in which the chief symptoms have been loss of appetite, diarrhea alternating with constipation, irritation of the kidneys, skin eruptions, and mental depression. Such results are said to have followed the use of articles of diet preserved by salicylic acid. The use of such foods is especially dangerous in the case of aged and very susceptible persons.

Fatal Dose.—Individuals differ greatly in their susceptibility to salicylic acid. Moreover, in most of the cases in which death occurred there was disease, and it is impossible to determine to what extent the fatal issue was due to the drug.[3] In one case a patient suffering from acute rheumatism died after taking something over 1 ounce (31 gm.) in four days.[4] Death has, however, been attributed to smaller doses. On the other hand, very large doses have been administered without poisonous symptoms appearing—for example, an ounce in twenty-four hours.

Treatment.—If the symptoms of irritation are not too marked, the stomach should be washed out with warm water. Milk and eggs may be administered, as in cases of poisoning with mineral acids.

Postmortem the lesions of gastritis, enteritis, and acute nephritis

[1] Benzoic acid, formaldehyd, and other food preservatives have partially replaced salicylic acid in recent years.

[2] *Bull. de l'Acad. de Méd.*, 22, Paris, 1886, vol. xvi., p. 583.

[3] See Goodhart, *Brit. Med. Jour.*, 1880, vol. i., p. 130; Philpot, *Lancet*, 1877, vol. ii., p. 636; *Bull. de l'Acad. de Méd.*, 2 s., 1877, vol. vi., p. 753.

[4] Quincke, *Berlin. klin. Wochenschr.*, 1882, vol. xix., p. 710.

have been found; marked hyperemia of all the organs is usually present.

Isolation.—After poisoning with salicylic acid the substance passes quickly into the urine, partly as the sodium salt and partly as salicyluric acid. In order to isolate the poison it is necessary only to acidify with sulphuric acid and shake out with ether or chloroform. Other tissues should be extracted with acidified water, and the aqueous extract made alkaline and evaporated to dryness on the water-bath. The residue is then acidified with sulphuric acid and extracted repeatedly with ether. On evaporation of the ether salicylic acid will remain in the form of crystalline needles.

Tests.—The reactions with ferric chlorid, Millon's reagent, and bromin water, which are described under Carbolic Acid, are equally applicable to the detection of salicylic acid. The following characteristic test, however, will easily distinguish between the two substances:[1]

A small amount of the suspected substance is heated in a test-tube with 1 c.c. of methyl-alcohol and half as much concentrated sulphuric acid. The mixture is allowed to cool and again heated, when, if salicylic acid be present, the odor of oil of wintergreen will be noticed.

Salol (phenyl salicylate, $C_6H_4.OH.COOC_6H_5$), a white powder, almost insoluble in water, is decomposed by the pancreatic juice into phenol (40 per cent.) and salicylic acid (60 per cent.). The use of salol in medicine has led to a few cases of poisoning. The symptoms are partly those of carbolic and partly those of salicylic acid poisoning; vomiting, fever, drowsiness, hematuria, roaring in the ears, etc., have been described. The urine is a dark olive green, as in phenol poisoning. In a case reported by Hesselbach[2] a woman with a contracted kidney and suffering from acute rheumatism died on the fifth day after the administration of 2 drams (7.76 gm.) in the course of eight hours; another case is reported in which death was attributed to 15 grains (1 gm.) of salol taken twelve days previously.[3]

Detection.—Salol suffers hydrolysis in the body, giving rise to salicylic acid and phenol, both of which can be found in the urine. The best method of identification is to show the presence of both acids after submitting the suspected substance to saponification.

PICRIC ACID.

Picric acid (trinitrophenol, $C_6H_2(NO_2)_3OH$) is a yellow, crystalline substance, sparingly soluble in water, and possessing a strong coloring power. Picric acid and the picrates are much used in the arts, especially as a dye and as a constituent of certain high explosives (melenite, lyddite, emmensite). It has been used for coloring foods and as an adulterant of beer. It has been used to a slight extent in medicine, especially in the treatment of skin diseases and burns.

[1] Curtman, *Zeitschr. f. anal. Chem.*, vol. xxvi., p. 641.
[2] Hesselbach, *Fortschr. der Med.*, 1890, p. 453; see Sahli, *ibid.*, p. 661.
[3] Chalpowski, *Therap. Monatsh.*, 1891, vol. v., p. 213.

Poisoning has resulted from the use of picric acid as a means of committing suicide;[1] in other cases workmen have been poisoned by handling it. Poisoning has also been caused by its absorption from the skin when applied in the form of a salve in skin diseases[2] and from the drinking of beer adulterated with it.

Picric acid is an irritant to the skin and mucous membranes. It precipitates albumin and so causes local necroses. It irritates the central nervous system and leads to convulsions. Like other nitro-bodies, it has a tendency to cause destruction of the red blood-corpuscles and to lead to the formation of methemoglobin.

Symptoms.—When the poison is taken internally, the symptoms[3] begin with pain in the abdomen, vomiting, and diarrhea. The urine is at first dark yellow, but later becomes of a red-brown color; it contains no bile. There may be anuria and strangury. The vomited matters and the feces are stained yellow. The conjunctivæ and later the skin assume a yellow color. Eczema and itching are common results. Prostration, stupor, and occasionally convulsions, followed by collapse, have been observed. The excretion of the poison is slow, and the yellow discoloration of the skin may persist for several days.

When poisoning has resulted from the external application of picric acid, as in industrial poisoning, and after its use in the treatment of skin diseases, intense itching and eczema have been prominent symptoms: stomatitis and gastro-intestinal symptoms have also resulted from some of the poison being carried to the mouth by the fingers. After the application of 7 grains (0.455 gm.) of picric acid to the vagina the skin became yellow; the urine was red, and somnolence and burning in the stomach were noticed.[4] A form of chronic poisoning has been noticed in laborers engaged in the manufacture of melinite: the symptoms were nasal catarrh, diarrhea, abdominal pains, and dizziness.

The **fatal dose** of picric acid is not known: from 15 to 30 grains (1 to 2 gm.) cause toxic symptoms, but recovery has followed the taking of a "coffeespoonful"[5] and also nearly 90 grains (6 gm.).[6]

The **treatment** should consist in thoroughly washing out the stomach. The bowels should be emptied by the use of suppositories. Purgatives are not to be recommended, owing to the irritation of the intestine caused by them. The white of egg and milk should be administered, as they form insoluble compounds with the poison. The administration of large doses of dextrose has been recommended, as this substance seems to aid the reduction of the picric acid to the less poisonous picraminic acid.[7]

Detection.—Extract the material under examination with alcohol, evaporate to a syrup, and shake out the picric acid with successive

[1] Karplus, *Zeitschr. f. klin. Med.*, 1893, vol. xxii., p. 210.
[2] Waldo, *Brit. Med. Jour.*, 1897, vol. i., p. 331.
[3] Adler, *Wien. med. Wochenschr.*, 1880, vol. xxx., p. 819.
[4] Cheron, *Jour. de thérap.*, 1880, vol. vii., p. 132.
[5] Halla, *Prag. med. Wochenschr.*, 1882, vol. vii., pp. 490, 503.
[6] Karplus, *loc. cit.*
[7] Rymsza, *Ein Beitrag z. Toxikol. der Pikrinsäure*, Dorpat, 1889.

often another absorption band is present.[1] The blood may contain but 1 per cent. of oxygen (normally it contains 17 per cent.), and is incapable of taking up more when shaken with air. This decrease in the oxygen-carrying power of the blood leads to a diminution of oxidation and to the appearance of abnormal products in the urine. Some of the nitrobenzene seems to be reduced to anilin, which also appears in the urine.

Symptoms.—When taken internally, there is a burning sensation, but other symptoms may not begin for from one-quarter to two or three hours. The patient may walk about, eat, and appear quite normal,[2] and then the symptoms begin quite suddenly and resemble to some extent those of hydrocyanic acid poisoning. The face becomes grayish or bluish-white, the lips and nails purple (these changes may precede the subjective symptoms); the pulse is weak and rapid; vision is disturbed; the gait is unsteady; the breath has the odor of bitter almonds; vomiting, dizziness, and headache follow, and finally coma comes on, often with great suddenness. Muscular twitchings and involuntary evacuation of the feces and urine may occur. Well-marked trismus, making the passage of the stomach-tube very difficult, has been observed a number of times. The temperature falls, and the respiration is frequently of the Cheyne-Stokes type. The pupils may be contracted or dilated, and do not react to light. The urine is dark, smells of nitrobenzene, and contains a reducing substance. Death usually occurs in coma from failure of the respiration; sometimes it results from the inspiration of vomited matters.

If the patient survives, recovery is somewhat slow; nausea and vomiting may persist; there is frequently a rise of temperature, often associated with necrotic processes, as in one case of the heels. Jaundice may appear in three or four days[3] and continue until death occurs several (*e. g.*, seventeen) days afterward. Ehlich and Lindenthal[4] found the blood in such a case to be very similar to that of pernicious anemia.

The symptoms following the inhalation of the vapor of nitrobenzene, the most frequent form of poisoning, are drowsiness, headache, unsteadiness of gait, the patient acting like a person intoxicated, and stupor. There are cardiac irregularity, loss of voluntary power, and extreme cyanosis. The urine is reddish-brown and has the odor of bitter almonds. Coma may come on very suddenly, with symptoms resembling those of apoplexy, death being preceded by Cheyne-Stokes respiration and occasionally by convulsions. Of 61 cases collected by Lewin, 24 (or 39.3 per cent.) died.

Men engaged in industries involving the use of nitrobenzene frequently suffer from a form of chronic poisoning[5] in which languor,

[1] These blood changes are constantly found in animals poisoned with nitrobenzene, but have been observed but seldom in man, owing, probably, to faulty technic; see, however, Ehlich and Lindenthal, *Zeitschr. f. klin. Med.*, vol. xxx., p. 427.

[2] Dodd, *Brit. Med. Jour.*, 1891, vol. i., p. 849.

[3] Schild, *loc. cit.*

[4] Ehlich and Lindenthal, *Zeitschr. f. klin. Med.*, vol. xxx., p. 427.

[5] White, *Proc. Med. Jour.*, 1892, vol. xi., p. 462.

A convenient method of effecting this reduction is as follows: A portion of the nitrobenzene is dissolved in warm dilute alcohol, some zinc-dust is added, and the material treated with the hydrochloric acid, a drop at a time, until there is a brisk evolution of gas. The reaction is allowed to proceed until the odor of nitrobenzene has disappeared. After the solution has been diluted with water and made alkaline, the anilin may be extracted with ether.

DINITROBENZENE.

Meta-dinitrobenzene ($C_6H_4(NO_2)_2$) is used in the manufacture of roburite, some kinds of rifleite, and other explosives. Poisoning, both acute[1] and chronic, has been caused by dinitrobenzene as well as by the explosives. The poison is usually absorbed by the skin.

Symptoms are very similar to those caused by nitrobenzene. A case[2] is reported in which a family of 7 were poisoned by dinitrobenzene contained in roburite which had been sprinkled on the floor and beds as an insecticide; the symptoms were vomiting, diarrhea, headache, pain in the abdomen, cyanosis, quick shallow respirations, rapid pulse, dulness of the mental faculties, and dark-colored urine. In the chronic form of poisoning amblyopia[3] has frequently been observed.

Detection.—Dinitrobenzene may be shown by reduction in the tissues with tin and hydrochloric acid to metaphenylenediamin, extracting this substance with ether, and testing a solution in dilute sulphuric acid with a nitrite (see test under Amyl Nitrite).

ANILIN.

Most of the cases of poisoning by "anilin" have been due not to pure anilin, but to "anilin oil," a mixture of anilin and toluidins. Most cases are "industrial" and are caused by the inhalation of the vapors by workmen engaged in the manufacture of the substance or of its derivatives; the poison is also readily absorbed from the skin. Occasionally the liquid is taken internally, as for the purpose of committing suicide.[4]

Properties.—Anilin (amido-benzene, phenylamin, $C_6H_5NH_2$) is a colorless, oily, inflammable liquid, of a peculiar odor and a burning, aromatic taste. It boils at 184° C. (363.2° F.). It is sparingly soluble in water, but readily soluble in alcohol and ether. It has basic properties and forms well-defined salts. The toluidins, so often associated with anilin, boil at about 200° C. (392° F.). The physiologic action of anilin is very similar to that of nitrobenzene—*i. e.*, it stimulates and then paralyzes parts of the central nervous system, but the chief effects are upon the blood.

[1] Spurgin, *Brit. Med. Jour.*, 1891, vol. i., p. 801.
[2] Monks, *Lancet*, 1902, vol. i., p. 89.
[3] *Medical News*, 1895, p. 76.
[4] Müller, *Deutsch. med. Wochenschr.*, 1887, vol. xiii., p. 27; Dehio, *Berlin. klin. Wochenschr.*, 1888, vol. xxv., p. 11.

Symptoms.[1]—The most marked symptom in anilin poisoning is a grayish-blue color of the face, lips, and mouth; in some cases this coloration, which seems to be due largely to the formation of methemoglobin, is almost the only effect. Other symptoms are headache, giddiness, weakness, staggering gait. In more severe cases there are great headache, somnolence, small, frequent pulse, and loss of consciousness. Convulsions may occur, and then coma and death. If the case is somewhat prolonged, painful micturition,[2] jaundice,[3] or diarrhea[4] may follow. When the poison is taken internally, vomiting may result. The breath has an odor resembling that of coal-tar. Complete recovery may not occur for five or six weeks.

Anemia, skin eruptions, various nervous symptoms, and amblyopia have been observed among men employed for some time in anilin factories, and have been attributed to chronic poisoning by anilin.

Fatal Dose and Period.—The fatal dose and period are not known. Twenty-five c.c. (7 fluidrams) of anilin oil caused the death of a young woman in twenty-four hours;[5] in another case recovery is said to have followed the taking of 75 c.c. ($2\frac{1}{2}$ fluidounces). In Dehio's case recovery followed the ingestion of 10 grams (about 2.75 fluidrams). Smith[6] reported a case in which a woman died twelve hours after taking 3 ounces (90 c.c.) of marking ink that consisted largely of anilin.

Treatment.—The patient should be brought into the fresh air, and if there is anilin on his clothes or hands, it should be removed. Stimulants should be administered. If the anilin has been taken per os, the stomach should be washed out and saline purgatives, stimulants, and diuretics administered; alcohol should not be given.

Postmortem Appearances.—The postmortem appearances are not characteristic. Methemoglobin is found in the blood, and the odor of anilin may be noticed in the organs.

Isolation.—The material under examination is made strongly alkaline and submitted to distillation with steam, when any anilin that is present passes over easily and will appear as colored oil-drops in the aqueous distillate. As the substance is soluble to some extent in water, oil-drops may not be observed, so that in any event the distillate is shaken out with ether, which, on evaporation, leaves the anilin as a more or less highly colored oil.

Anilin is the mother substance from which a large number of derivatives known as "anilin dyes" have been prepared. Many of these colors can be produced in solutions of extreme dilution, and have, therefore, been utilized as analytic tests.

Tests.—1. On the addition of a small quantity of a solution of bichromate of potassium to a drop of concentrated sulphuric acid that

[1] Knaggs, *Med. Times and Gaz.*, 1862, vol. i., p. 583; Mackenzie, *ibid.*, 1862, vol. i., p. 239; Smith, *Lancet*, 1894, vol. i., p. 89.

[2] Starck, *Therap. Monatsh.*, 1892, p. 376.

[3] Dehio, *loc. cit.*

[4] Franck and Beyer, *Münch. med. Wochenschr.*, 1897, vol. xliv., p. 57.

[5] Müller, *loc. cit.*

[6] Smith, *loc. cit.*

contains a trace of anilin a blue color slowly appears, which persists for many minutes and finally disappears. In the presence of more or less water the color is either green or black, according to the concentration, but under no conditions does the blue color pass rapidly through purple into red. This reaction for anilin, which is conveniently made on a white porcelain surface, involves the same reagents as are used in the principal color reaction for strychnin, and in trials for poisoning by strychnin the analyst is sure to be annoyed with senseless questions concerning anilin, questions that could be asked only by a person who had never performed the reaction with both substances.[1]

2. On the addition of a solution of calcium hypochlorite to a solution of free anilin a violet color is produced, which, under certain conditions of concentration, passes through the shades of purple into a dirty red. With a solution as dilute as 1 : 10,000 the violet color is pale or does not appear at all, but on the addition of a few drops of very dilute ammonium sulphid a rose-red color is produced, even when the original solution contained only 1 part of anilin in 250,000 parts of water.

3. The isonitrile reaction given under Chloroform serves just as well for the detection of anilin.

4. Anilin salts in great dilution yield with bromin water a flesh-colored precipitate of tribromanilin.

ANILIN AND SIMILAR DYES.

Numerous cases of poisoning of greater or less severity have resulted from the wearing or handling of articles dyed with anilin dyes or from the eating of food (sausages, confectionery, etc.) colored by them. Workmen who manufacture or make use of these dyes have also suffered from their effects. The effects have been partly local (eczema, edema of the face and hands, vomiting) or general (anesthesias, etc.). In some cases the poisonous substance has been anilin oil, as when a number of children were poisoned by wearing boots dyed with anilin-black, which contained 90 per cent. of anilin oil.[2] In other cases impurities have been the cause of the poisonous effects; thus fuchsin, of itself almost inert, is (or was) often contaminated with arsenic, which produces its characteristic effects upon persons who work with fuchsin, or who take it in food or in wine colored by it. In other cases the impurities have been carbolic acid, free anilin, toluidin, etc. The same holds true for such dyes as anilin-brown, methyl-violet, malachite-green, the rosolic acids, eosin, etc. Sometimes lead or cadmium is present as an impurity. Sometimes the injurious effects of clothes dyed with these colors have been due to the incomplete removal of the excess of the "mordants" (*i. e.*, substances with which the cloth was treated in order to make it combine with the dye) or of sulphuric acid. In other cases the dyes themselves seem to be poisonous; thus

[1] Beissenhirz, *Liebig's Annalen*, vol. lxxxvii., p. 376.

[2] Bronardel, *Ann. d' hyg. pub.*, 1900, pt. ii.; see also Lament and Guillemin, ***Jour. des Praticiens***, 1901, p. 131.

gloves dyed with chrysoidin or Bismarck brown have caused severe eczema. Frequently the local effects seem to be due to the excessive use of soda or of bleaching powder for the purpose of removing stains from the hands.[1]

Dyes somewhat similar to the above are contained in some of the "hair restorers," and have caused very severe symptoms when applied to the hair.[2] Methylene-blue, which has been somewhat extensively used in medicine, causes, in large doses or after long-continued use, diarrhea, headache, dizziness, and delirium. Safranin is very poisonous when applied to the injured skin or subcutaneously.[3] Anilin orange, often used in dying feathers, has caused severe local effects upon the hands of those working with it. White[4] reports cases of eczematous dermatitis of the neck and face produced by wearing fur tippets colored by some dark dye.

It may be said in general, however, that most of these dyes are mixtures and that it is impossible, in most instances, to determine how injurious the chemical individuals contained in them are. The toxicity of most of the coal-tar dyes has been much exaggerated;[5] if free from poisonous contaminations, they are for the most part harmless, at least in the quantities ordinarily employed. Such dyes are often used, however, to give a natural color to sausages and other articles of food that have deteriorated or to conceal excess of fat, tendon, etc.;[6] hence all articles of food colored in this way are justly regarded with suspicion.

Many nitrobodies used as dyes are unquestionably poisonous. Thus the potassium and sodium salts of dinitrocresol, which are used as substitutes for saffron under the names of "Victoria yellow," "Victoria orange," or anilin-orange, are toxic; a woman died from 75 grains (4.88 gm.) of one of these preparations, which she had taken instead of saffron with the object of bringing on an abortion.

ACETANILID AND SIMILAR COMPOUNDS.

Many derivatives of anilin have been introduced into medicine as antipyretics and analgesics, and some of them have caused numerous cases of poisoning. The oldest of these drugs, introduced in 1886, is acetanilid ($C_6H_5.NH.COCH_3$), a substance formed by substituting one H atom of the amid group of anilin by acetyl. Exalgin ($C_6H_5.NCH_3$-$COCH_3$) differs from acetanilid only in having a methyl group in the side chain. Phenacetin ($C_6H_4<\begin{matrix}NH(COCH_3)\\OC_2H_5\end{matrix}$) differs from acetanilid in having an ethoxyl group in the para position. Similar compounds are lactophenin, malakin, phenocol, etc. All these substances cause a fall of temperature in fever; this action is probably due to the formation

[1] Blaschka, *Deutsch. med. Wochenschr.*, 1891, vol. xvii., pp. 1241, 1265.

[2] Puppe, *Vierteljahrsschr. f. ger. Med.*, 1896, 3. Folge, vol. xii., p. 116 (Suppl.) (paraphenylendiamin).

[3] Weyl, *Zeitschr. f. Hygiene*, 1889, vol. vii., p. 37.

[4] *Boston Med. and Surg. Jour.*, 1902, vol. cxlvi., p. 269.

[5] See Georgievics, *Lehrb. der Farbenchemie*, 1895, p. 10.

[6] *Das Sanitätswesen des Preuss. Staates*, 1895–97, pp. 446 and 477.

of simple derivatives of para-amido-phenol ($C_6H_4<^{NH_2}_{OH}$) in the tissues. This decomposition takes place rapidly with some, slowly with other, of the substances.

The **physiologic action** of these substances is very complex and but little understood; their effects in disease are different from those in health. Moreover, individuals differ greatly in their susceptibility to their influences. The most constant and characteristic effect produced by large doses is an alteration of the red blood-corpuscles. This alteration consists in the formation of methemoglobin, which may be detected by the spectroscope.[1] With small doses the corpuscles remain intact, but large doses destroy the cells and free the methemoglobin. This action leads to the appearance of marked cyanosis, and the excretion of the altered blood pigment leads to nephritis and the appearance of blood pigments and of blood in the urine. If the effects are severe and the patient lives for some time, jaundice is not uncommon. In milder cases of poisoning marked cyanosis is sometimes present, although methemoglobin cannot be detected in the blood; the cause of the cyanosis in these cases has not been satisfactorily explained.

Most of these bodies possess some antiseptic power and have been used to some extent in dressings to wounds. Symptoms of general poisoning have followed the absorption of the drug from wounds so treated,[2] but most cases of poisoning have resulted from their internal administration as antipyretics. In a few cases they have been taken with suicidal intent. They are frequently contained in "headache powders," and death has resulted from the excessive use of these preparations.

Acetanilid (*Acetanilidum*, U. S. P., antifebrin) is the most important of this series from the toxicologic standpoint. It is a colorless, crystalline body, insoluble in water, but soluble in alcohol, ether, and chloroform.

The chief **symptom** caused by acetanilid is marked and long-continued cyanosis; in mild cases of poisoning this is the only symptom. In severe cases unconsciousness, followed by marked restlessness, vomiting, and dyspnea, occurs. The pulse is rapid and irregular; there are often profuse perspiration and then collapse. Various forms of skin eruptions and also convulsions and delirium have been described.

In a fatal case described in detail by Brown[3] there were a general hyperesthesia and increased reflex actions. There were persistent vomiting and slight delirium. There was first an elevation, later a depression, of the temperature. There was at first extreme cyanosis. One of the most marked effects was a progressive destruction of the red blood-corpuscles; on the eighth day (the day of death) they were 1,116,000, with 30 per cent. of hemoglobin. There was marked leukocytosis

[1] Muller, *Deutsch. med. Wochenschr.*, 1887, vol. xiii., p. 28.
[2] Menasses, *Internat. Med. Mag.*, 1901
[3] *Amer. Jour. Med. Sci.*, 1901, vol. cxxii., n. s., p. 770.

(66,450), and a large number of nucleated red cells of all sizes was present. There were hematoporphyrinuria, beginning twenty-four hours after the dose was taken, and complete suppression of urine three days before death. There were acute diffuse nephritis, hemorrhage from the intestines, and acute progressive jaundice.

The long-continued use of medicinal doses leads to a form of chronic poisoning in which cyanosis, dyspnea, anemia, and loss of weight are common symptoms.

The **fatal dose** of acetanilid is not known. Recovery has followed the taking of 1 ounce (31.1 gm.), while in other cases death seems to have occurred after a few doses of from 8 to 30 grains (0.52 to 1.95 gm.). In Brown's case death occurred eight days after taking 60 grains (3.9 gm.) in the course of a few hours. Severe symptoms of poisoning have followed the administration of less than 2 grains (0.13 gm.) to women suffering from typhoid fever.[1] A form of chronic poisoning may result from the long-continued use of secret remedies containing acetanilid.[2]

The **treatment** consists in thoroughly washing out the stomach, and in administering saline purgatives and cardiac stimulants (strychnin, digitalis, etc.). If the cyanosis and dyspnea are severe, bleeding, followed by the infusion of normal saline solution, is recommended.

Detection.—The material under examination is extracted with strong alcohol for the removal of proteids. After evaporating the alcohol the residue is taken up in acidified water, and the acetanilid is shaken out of the acid fluid with ether or chloroform. To the residue obtained after evaporating the organic solvent the following tests may be applied:

1. Upon warming a mixture of acetanilid, chloroform, and alcoholic solution of potassium hydroxid the offensive odor of isobenzonitrile can be detected.[3]

2. Heat about 0.1 gram of the suspected substance for a minute with 1 c.c. of hydrochloric acid, and add a small quantity of a solution of calcium hypochlorite and phenol. A red color is produced, which changes to a fine blue on the addition of ammonia.[4]

For other reactions and proof in the urine see Vulpius.[5]

Phenacetin, although a safer antipyretic than acetanilid, occasionally gives rise to cases of poisoning; in rare cases death seems to have resulted from its use.[6] The symptoms are similar to those described under acetanilid, and consist in marked cyanosis, changes in the blood, dizziness, sleepiness, profuse perspiration, etc.

[1] Sembritzki, *Therap. Monatsh.*, 1889, vol. iii., p. 267.
[2] Dimsey, *Brit. Med. Journ.*, 1896, vol. ii., p. 195; see also pp. 89, 653.
[3] Kottmayer, *Chem. Centralbl.*, 1890, vol. ii., p. 1030.
[4] *Zeitschr. f. anal. Chem.*, vol. xxvii., p. 666. [5] *Ibid.*, vol. xxviii., p. 103.
[6] Krönig, *Berlin. klin. Wochenschr.*, 1895, vol. xxxii., p. 998.

ANTIPYRIN.

Antipyrin was introduced into medicine as an antipyretic in 1884, and has caused many cases of poisoning; a number of deaths have been attributed to its use.[1] Practically all the cases of poisoning have been medicinal; in some cases an overdose was given, but in many of them the unfavorable symptoms seem to have been dependent upon some abnormal or unusual condition of the patient.

Properties.—Antipyrin (phenyl-dimethylpyrazolon, $C_6H_5N.CO.CH.NCH_3.C.CH_3$) occurs in the form of colorless, inodorous crystals, with a bitter taste, very soluble in water, alcohol, and chloroform. The physiologic action of antipyrin is very similar to that of acetanilid.

Symptoms.—Probably the most frequent result of poisoning by antipyrin has been the appearance of various forms of skin eruptions and of edema, especially of the face.[2] Among other effects are dyspnea, cardiac weakness, coma, and collapse. There is usually cyanosis, but this is less marked than with acetanilid. Muscular pains, trembling, and twitching of an epileptiform character have also been described. Occasionally amblyopia and ringing in the ears have been observed. Death in collapse has occurred a number of times after the administration of antipyrin, but in most of these cases it is doubtful whether the fatal issue was due to the drug or to the condition of the patient.[3] Occasionally death has been preceded by convulsions.

Long-continued use of antipyrin has led to a form of chronic poisoning in which skin eruptions, dyspepsia, loss of appetite, drowsiness, and muscular weakness have been prominent symptoms.

The **fatal dose** of antipyrin is not known. Death has followed, within a short time, the administration of 22 grains (1.43 gm.) to a consumptive and of 15 grains (1 gm.) to patients suffering with angina pectoris.[4]

The **treatment** is the same as in cases of poisoning by acetanilid.

Detection.—The isolation of the substance from the tissues is easily accomplished by preparing an acid aqueous extract free from proteids, as in an examination for alkaloids.[5] The acid solution is freed from various impurities by shaking with petrolic ether, and the antipyrin finally removed with chloroform. Upon evaporation of the chloroform antipyrin will remain in a form sufficiently pure for the following tests:

1. Aqueous solutions of antipyrin produce a red color with ferric chlorid solution, which is discharged by mineral acids.[6]

2. To a small quantity of an aqueous solution of antipyrin is added a drop or two of a dilute solution of potassium nitrite acidified with sulphuric acid. The characteristic green color of nitroso-antipyrin is

[1] See Lewin, *Die Nebenwirkungen der Arzneimittel*, 1899, p. 459.

[2] See cases reported by Jennings, *Lancet*, 1888, vol. i., p. 364; Short, *Brit. Med. Jour.*, 1892, vol. i., p. 1253; Seiler, *Therap. Monatsh.*, 1902, vol. xvi., p. 659.

[3] Barrs, *Lancet*, 1885, vol. i., p. 382.

[4] Lewin, *loc. cit.*

[5] Consult p. 331 *et seq.* in section on General Principles of Toxicology.

[6] Blumenthal, *Jahresber. d. Chem.*, 1886, 1893.

produced. The reaction serves for the demonstration of 1 part of antipyrin in 10,000 parts of water.[1]

CANTHARIDES.

Poisoning with cantharides is not common; most of the cases reported have been due to the use, by the laity, of the substance as an aphrodisiac or as an abortifacient, or have occurred through accident.[2] It has, however, been used for the purpose of murder. A few years ago it was proposed to treat tuberculous affections with cantharidinates, and a number of cases of poisoning resulted from this practice. Severe poisoning[3] and even death have resulted from its absorption from the skin, to which it had been applied as a vesicant.

Properties.—Cantharides (*Cantharis*, U. S. P., Spanish fly) is the dried Coleopterous beetle, *Cantharis vesicatoria*. When ground, cantharides forms a grayish-brown powder containing shining green particles. The active principle is cantharidin ($C_{10}H_{12}O_4$), the anhydrid of cantharidic acid. The powdered drug or the tincture has usually been used, but a number of cases of poisoning have resulted from the use of the salts of the acid.

Symptoms.—The symptoms are due largely to the local irritant action of the poison; this action is exerted at the point of application or at the point of excretion of the drug. Applied to the skin, cantharides or cantharidin produces redness and pain, followed by vesicles, which later coalesce to form a blister. Gangrene may result from its prolonged application to the skin. Some of the drug may be absorbed[4] and excreted along the alimentary and urinary tracts, and thus cause the same symptoms as when the drug is taken internally.

Taken internally,[5] cantharides produces the same changes on the mucous membrane as on the skin. Vesicles form, and there is an intense burning pain in the esophagus and stomach; there may be intense thirst, but also inability to swallow. Vomiting usually occurs; the vomited matter may contain blood and shreds of mucous membrane. Bloody diarrhea with the most intense abdominal pain follows. There are weakness and a state of collapse, and death may occur in from sixteen to twenty hours after the poison was taken.

In less acute cases the symptoms arise from the organs in which the drug is excreted—viz., the alimentary and genito-urinary tracts.[6] The symptoms arising from the latter organs are irritation of the bladder with a constant desire to urinate, strangury, acute nephritis with albuminuria, and sometimes hematuria. The inflammation of the bladder and urethra produces pain and frequently leads to priapism. In pregnant women abortion has occurred. The genital organs of both male and female are swollen and inflamed, and there is frequently, although

[1] Knorr, *Liebig's Annalen*, vol. ccxxxviii., p. 212.
[2] Revilliout, *Gaz. des Hôp.*, 1880, vol. liii., p. 698.
[3] Bressler, *Therap. Gaz.*, 1890, p. 150.
[4] Bressler, *loc. cit.* [5] See Jefferiss, *Brit. Med. Jour.*, 1876, vol. i., p. 190.
[6] Clarke, *Lancet*, 1881, vol. i., p. 499.

by no means always, increased sexual desire. The nephritis may lead to death.

In some cases there are severe nervous symptoms, with dyspnea, convulsions,[1] trismus, and tetanus; some of these symptoms may result indirectly from the action of the poison upon the alimentary and urinary tracts.

Fatal Dose.—The actual dose of cantharides necessary to cause death is not known. Twenty-five grains (1.63 gm.) of the powdered drug have caused death; in another case recovery followed the ingestion of 42 grains (2.7 gm.).[2] A boy of seventeen died fourteen days after taking an ounce of the tincture. A single fly which was swallowed by a child caused distressing symptoms. The fatal dose of cantharidin is estimated to be from $\frac{1}{10}$ to $\frac{1}{3}$ of a grain (0.007 to 0.022 gm.).

Fatal Period.—Death does not usually occur until several hours or days (one to fourteen) after the poison has been taken; in the cases that are rapidly fatal the chief symptoms arise from the alimentary tract; in the less acute cases death usually results from nephritis.

Treatment.—If the esophagus and stomach are not too severely inflamed, the stomach should be washed out with warm water; otherwise it may be emptied by hypodermic injections of apomorphin. Mucilaginous drinks and opiates should be given to allay the pain; oil should not be used, as the cantharidin is readily soluble in it. The nephritis should be treated by administering large drafts of water and the use of diuretics (tea, etc.). The irritation of the bladder is relieved by washing this organ out with warm water.

The chief **postmortem appearances** are severe gastritis, enteritis, and nephritis; cystitis and urethritis have also been observed. Vesicles may be found in the mouth and pharynx. The contents of the stomach and intestines usually contain some remnants of powdered cantharides if the powder itself has been taken; the green, shining particles are very characteristic of the drug.

Detection.—The material under examination is heated with 8 per cent. solution of potassium hydroxid until a perfectly homogeneous mass is obtained. The fluid is allowed to cool, extracted with chloroform for the removal of various impurities, and after acidification with sulphuric acid is treated with four times its volume of 95 per cent. alcohol. The product is boiled and filtered while hot. After cooling, the liquid is again filtered and the alcohol distilled off. The residual aqueous fluid is then shaken with chloroform, which takes out cantharidin.[3] The residue obtained by evaporation of the chloroform may be recognized by its ability to blister the skin. There are no characteristic chemical tests.

DIGITALIS.

By digitalis (U. S. P.) is meant the powdered leaves of *Digitalis purpurea*, or common European foxglove, a plant that is now cultivated

[1] Sedgwick, *Med. Times and Gaz.*, 1864, vol. ii., p. 617.
[2] Beck, *North Amer. Practitioner*, 1891, vol. iii., p. 522.
[3] Dragendorff, *Ermittelung von Giften*, Gottingen, 1895, p. 322.

or is found growing wild in parts of the United States. Digitalis and its preparations, especially the tincture and the infusion, are used very extensively in medicine as cardiac stimulants; certain bodies reputed to be the active principles are used to a more limited extent. As a matter of fact, there is still much doubt as to what the active principles of digitalis are. An extremely toxic glucosid, digitoxin, occurs in the leaves, and the activity of the preparations made from these has usually been attributed to this body. Recently Kiliani[1] has obtained from the leaves a body that is soluble in water and that seems to be identical with the digitalein obtained several years ago by Schmiedeberg. The leaves also contain a glucosid, digitophyllin, and another glucosid resembling digitalin; digitalin itself is probably absent. The seeds contain digitalin and a very small amount of digitoxin. Both leaves and seeds contain the saponin-like and almost inert body, digitonin, in considerable quantities. The "digitalins" of commerce are usually mixtures of some of these bodies and certain of their decomposition-products. They vary greatly in physiologic activity, some being almost inert while others are very toxic.

FIG. 66.—Foxglove, or digitalis (*Digitalis purpurea*).

Nearly all the reported cases of poisoning by digitalis have been accidental, and most of these have been due to the administration of too large medicinal doses. It was used for the purpose of murder in a very famous French case.[2]

Symptoms.[3]—The symptoms following the administration of digitalis or of one of its preparations have varied in different cases. Sometimes the symptoms have begun within an hour; in other cases, especially in those in which the digitalis was taken in the form of hard, not easily soluble, pills, they were delayed for a day or two. One of the earlier symptoms is a marked slowing of the heart: the pulse may fall to 40 or even to 25 a minute. At the same time the force of

[1] Kiliani, *Arch. d. Pharm.*, 1899, vol. ccxxxviii., p. 464.

[2] Tardieu and Roussin, *Gaz. des Hôp.*, 1864, p. 330; an abstract of this case is given in Maschka's *Handbuch d. Vergift.*, 1882, p. 495.

[3] See, for example, Mawr, *Lancet*, 1880, vol. i., p. 166 (case of a woman who recovered after taking nearly 1 grain (0.065 gm.) of Homolle's digitalin in the form of pills).

the beat is greatly increased, and the patient may suffer from throbbing of the carotids. There is great irritation of the stomach, leading to nausea and very persistent vomiting, abdominal pains, and thirst; sometimes there is diarrhea. The urine is often suppressed. There are also various nervous symptoms, such as roaring in the ears, disturbances of vision, vertigo, violent headache, hallucinations, mild delirium, great prostration, and, frequently, twitchings of the muscles; these symptoms are especially marked after muscular exertion. Later the heart becomes rapid and irregular; dyspnea and collapse follow, and finally death in coma, or sometimes in convulsions. In a number of cases death has occurred suddenly from failure of the heart.

If the patient does not die, recovery is very slow and often interrupted by severe relapses. There are often attacks of dizziness, fainting, and irregularity of the heart.

In light cases of poisoning the chief symptoms are slowing of the heart, with nausea and vomiting.

There is a form of subacute poisoning in which the symptoms do not appear until the drug has been given for some time; this is the so-called cumulative form of digitalis poisoning. In the oft-quoted case of Köhnhorn,[1] a man, aged twenty-two, took, in order to escape military service, about 13 grains (0.85 gm.) of digitalis in the form of pills daily. On the eleventh day he had marked symptoms of gastro-intestinal catarrh and a pulse-rate of 52 a minute. He continued taking the pills until death, from sudden failure of the heart, took place on the twenty-eighth day.

The **fatal dose** of digitalis and its preparations is not known; 36 grains (2.34 gm.) of the powdered leaves have caused death, while recovery has followed the taking of 1 dram (3.9 gm.). Death has been caused by 1 ounce (30 c.c.) of the tincture.[2] Of the various constituents of digitalis, digitoxin is unquestionably the most toxic; in the well-known case of Koppe,[3] $\frac{1}{30}$ of a grain (0.002 gm.) of digitoxin caused most severe poisoning, and it is very probable that $\frac{1}{15}$ of a grain (0.004 gm.) would ordinarily be fatal. The commercial digitalins consist of digitoxin, digitalin, and other bodies in varying proportions, and it is impossible to state the fatal dose. A child, aged one year and eleven months, was very severely poisoned by about $\frac{1}{50}$ of a grain of Nativelle's digitalin,[4] and in another case $\frac{1}{30}$ of a grain of a similar preparation caused dangerous symptoms in a boy of fifteen; it is probable that $\frac{1}{15}$ of a grain and upward of most of these preparations would be very dangerous to an adult. There is, however, a preparation upon the market known as "German digitalin," which is given in doses of from $\frac{1}{10}$ to $\frac{1}{2}$ of a grain (0.007 to 0.032 gm.); it is said to contain digitonin (to the extent of 50 or 60 per cent.), digitalein, and small quantities of digitalin and digitoxin.

[1] Köhnhorn, *Vierteljahresschr. f. ger. Med.*, 1876, vol. xxiv., p. 278; see also *Lancet*, 1876, vol. i., p. 582.
[2] Rames, *Gaz. des Hôp.*, 1876, vol. xlix., p. 756.
[3] Koppe, *Arch. f. exper. Path. u. Pharm.*, 1874, vol. iii., p. 289.
[4] Radcliffe, *Brit. Med. Jour.*, 1901, vol. i., p. 338.

The **fatal period** is usually long, death seldom occurring for a day or two; it is frequently prolonged for several (five to thirteen) days.

The **treatment** consists in thorough evacuation of the stomach, the administration of stimulants (caffein, strychnin, and atropin), and the application of heat. Absolute rest in bed is very important.

There are no characteristic **postmortem appearances** after digitalis poisoning; occasionally there are some indications of gastritis.

Detection.—An aqueous extract of the suspected material is decolorized with animal charcoal and treated with neutral lead acetate as long as the reagent produces a precipitate. The filtrate from this precipitate is then treated with a solution of lead acetate and ammoniacal alcohol. This precipitate is filtered off, supended in water, and decomposed with sulphureted hydrogen. The digitalein will be found in the liquid, while digitalin remains with the lead sulphid and can be removed by extraction with chloroform. On evaporation of the chloroform digitalin will remain in crystalline form.[1]

Digitalin may be removed from alcoholic or aqueous solutions by agitation with freshly precipitated lead hydroxid. The lead precipitate assumes a flesh color on treatment with concentrated sulphuric acid, and the product becomes emerald green upon the subsequent addition of bromin.

It is very important to apply the physiologic test[2] to the suspected material; this is best done, if the amount of material is small, with frogs. The heart is exposed, the beats counted, and then 1 or 2 milligrams of the suspected material, in solution, is injected into a lymph-space. If the material belongs to the digitalis series, the heart will become slower, will empty itself more completely at each systole, and dilate less during diastole until it finally stops in systole. Of course, it is necessary carefully to control this experiment by similar ones carried out under the same conditions with known quantities of digitalin.

Strophanthus.—Strophanthus, the physiologic action and uses of which are very similar to those of digitalis, occasionally causes severe and even fatal[3] cases of poisoning; the symptoms are similar to those of digitalis poisoning, but the course is more rapid.

Isolation.—An acid aqueous extract of the material under examination is prepared as in an examination for alkaloids. The aqueous fluid is freed from various impurities by shaking with benzene, and the strophanthin finally removed by shaking with amyl-alcohol.

Tests.—1. Strophanthin yields a red color when treated in turn with sodium nitroprussiate solution and an alkali.

2. An aqueous solution of the suspected substance is treated with a trace of ferric chlorid and then with concentrated sulphuric acid. In the presence of strophanthin a brown precipitate is formed which, after an hour or so, becomes emerald green.

3. Strophanthin dissolves in 75 per cent. sulphuric acid with a fine

[1] Palm, *Zeitschr. f. anal. Chem.*, vol. xxiii., p. 22. [2] See Tardieu and Roussin, *loc. cit.* [3] See *Berlin. klin. Wochenschr.*, 1888, vol. xxv., p. 115.

violet color. This reaction will easily show the presence of half a milligram of the poison.

4. Strophanthin arrests the heart in systole, a phenomenon that can be brought about in frogs with $\frac{1}{100}$ of a milligram of the substance.

Oleander.—The common ornamental shrub, *Nerium oleander*, contains two or more active principles belonging to the "digitalis series"; a number of cases of poisoning, especially among children, are reported. The symptoms[1] have been vomiting, abdominal pains, vertigo, convulsive movements, insensibility, small, very slow pulse, and, in some cases, epileptiform convulsions, followed by coma and death. In cases that have recovered the pulse-rate has remained as low as 40 a minute for five days.

ERGOT.

Ergota (U. S. P.), ergot of rye, is the sclerotium of the parasitic fungus, *Claviceps purpurea*, replacing the grain of rye. It is also found on other grains, as on some native grasses of the West, where its ecbolic properties are well known to many tribes of Indians.

Most cases of poisoning by ergot have been due to the eating of bread made of diseased grain; such cases are now rare. Some cases of poisoning result from its use in medicine or from its use as an abortifacient.[2]

Properties.—Notwithstanding the fact that a great deal of work has been done upon the subject, the chemistry of ergot is in a very unsatisfactory condition. A large number of bodies have been described as occurring in it, and some of these have been used to a limited extent in therapeutics. Among the earlier of these preparations may be mentioned "ergotinin" and "ecbolin." It is customary at present[3] to ascribe the action of ergot to three imperfectly isolated bodies—ergotinic acid, cornutin, and sphacelotoxin. Ergotinic acid causes depression and paralysis of the spinal cord. Cornutin is probably a mixture of poisons, the physiologic action of which is to cause a stimulation of the medulla, which leads to convulsions, vomiting, purging, and a rise of blood pressure. Cornutin also causes wave-like contractions of the uterus. Sphacelotoxin is a resinous body that leads to irritation of the alimentary tract, causing vomiting and other abdominal symptoms. The most characteristic effect of sphacelotoxin, however, is the production of a dry gangrene; this gangrene seems to be due to a prolonged contraction of the arterioles, shutting off the blood-supply of the part and leading to a hyaline formation in the lumen of the vessels. Sphacelotoxin induces abortion in pregnant animals.

The official preparation commonly used is the fluid extract, the dose of which is 1 or 2 fluidrams (3.7 or 7.4 c.c.).

[1] See *Therap. Gaz.*, 1888, p. 452.

[2] Lincoln, *Toledo Med. and Surg. Jour.*, 1878, vol. ii., p. 243; *Pharm. Jour. and Trans.*, 1878, 3 s., vol. ix., pt. i., p. 541.

[3] Cushny, *Pharmacology and Therapeutics*, 1901, p. 460; see Santesson, *Skand. Arch. f. Physiol.*, 1902, vol. xiii., p. 107 (on "Cornutin Keller").

Symptoms.—Two forms of ergot poisoning are recognized, acute and chronic.

Acute poisoning by ergot may be caused either by the eating of bread made from diseased grain or by preparations of the drug taken internally.[1] The symptoms, which may not appear for some hours, usually begin with vomiting; there are burning pains in the abdomen and tingling of the extremities, great thirst, weakness, and diarrhea. There is frequently swelling of the face and extremities. The pulse is slow and weak. Abnormal sensations develop in the skin, especially in that of the hands and feet; there are feelings of chilliness and, later, twitchings or tonic contractions of the muscles. Ataxia, epileptiform convulsions, and mania have been observed. These symptoms are followed by a fall of temperature, and in many cases gangrene of the skin has been observed. Suppression of the urine, great prostration, coma, and death from failure of the respiration and heart may follow. Death may occur in a few hours or be delayed for a few days. If the patient recovers, abnormal sensations may persist for several days; swallowing may be difficult and painful. Sometimes a cataract forms in one or both eyes.

If the patient is pregnant, and especially if there is a predisposition to miscarriage, abortion, followed by severe hemorrhage, may occur; both mother and child may die. These results are especially liable to occur in the late stages of pregnancy; in fact, many consider it doubtful whether ergot has any influence upon the uterus during the first half of pregnancy. Most obstetricians consider the administration of ergot during parturition before the delivery of the child to be fraught with great danger to the child. Powerful contractions of the uterus may be set up by ergot if there are fibroids in the uterus.[2]

Fatal Dose and Period.—Ergot and the preparations of ergot vary so greatly in strength that it is impossible to state the fatal dose. Thirty grains (1.95 gm.) have caused severe poisoning,[3] but recovery has followed 150 grains (9.72 gm.). Gangrene and death are said to have followed 12 grains (0.77 gm.). One fluidram (3.75 c.c.) of the fluid extract caused great sleepiness, swelling and redness of the feet, and violent prickling of the extremities;[4] yet ounce doses have often been given without causing any poisonous symptoms. Death has occurred within one to three days after the appearance of the first symptoms. Death is more likely to follow the long-continued use of small[5] or medicinal doses than after one large dose.

The **treatment** consists in washing out the stomach and emptying the bowels by enemata or purgatives, such as calomel or castor oil. In collapse, warm baths with cold affusions are recommended; also

[1] Oldright, *Canadian Med. Jour.*, 1870, vol. vi., p. 404; Meadows, *Med. Times and Gaz.*, 1879, vol. ii., p. 397; Hulme, *Medical News*, 1887, vol. li., p. 538; Débierre, *Bull. de Thérap.*, vol. cvi., p. 52.

[2] Meadows, *loc. cit.*

[3] Meadows, *loc. cit.*

[4] Faulkner, *New York Med. Jour.*, 1884, vol. xxxix., p. 668.

[5] Davidson, *Lancet*, 1882, vol. ii., p. 526.

stimulants, such as coffee, tea, etc. If gangrene sets in, the parts should be bathed with warm water and wrapped in cotton.

Postmortem, ecchymoses in the stomach, intestines, kidneys, uterus, lungs, and peritoneum have been frequently observed. Microscopic examination sometimes reveals the presence of polyneuritis and inflammation of the arteries.

Chronic Poisoning.[1]—Chronic poisoning by ergot has nearly always been due to the eating of bread made from diseased grain; wide-spread epidemics of "ergotism" occurred in Europe during the Middle Ages, but are now rare. More rarely the medicinal use of ergot has led to chronic poisoning.

There are two distinct forms of ergotism—the spasmodic and the gangrenous types. In some epidemics both forms are present, but, as a rule, one form is much more prevalent than the other. The early symptoms are the same in each, and consist in indefinite pains, disturbances of digestion, etc. In spasmodic ergotism there is anesthesia of the fingers and toes, then of the extremities; later there are tetanic spasms of the muscles, followed, if the patient recovers from the first effects, by contractures of the limbs. Mental weakness or dementia is a not uncommon result. In the gangrenous form of ergotism there is acute pain in one or more limbs; the limb swells, is dark in color, and is very cold. Dry gangrene sets in, and fingers, toes, and even arms and legs and nose fall off; there is little pain or hemorrhage. Gangrene of internal organs may occur; thus cataracts are common. All these effects seem to be due to sphacelotoxin, which causes prolonged contraction of the vessels, followed by hyaline thrombosis.

Detection.—Dragendorff[2] found it impossible to identify ergot by the isolation of any of its active constituents from animal tissues. The substance yields trimethylamin when treated with alkalis. This property, however, is common to many organic compounds and, therefore, has little toxicologic value.

COTTON-ROOT BARK.

Gossypii Radicis Cortex (U. S. P.) has long had the reputation, among the negroes of the South, of being an abortifacient; it is used to some extent as an emmenagogue. Severe poisoning was caused by 4 ounces (120 c.c.) of the fluid extract taken for the purpose of producing abortion.[3]

MALE FERN.

Male fern (*Aspidium*, U. S. P., the rhizome of *Dryopteris Filix-mas* and of *D. marginalis*) yields a resinous body that is official under the name of *Oleoresina Aspidii*. The active principles of this resin seem

[1] See Allbutt's *System of Medicine*, vol. iii., p. 793; Kobert, *Arch. f. exper. Path. u. Pharmakol.*, vol. xviii., p. 346.
[2] *Ermittelung von Giften*, Göttingen, 1895, p. 294.
[3] Blakeley, *Med. News*, 1896, vol. lxviii., p. 416.

to be a number of neutral and acid bodies, the most important of which are aspidin, aspidinin, and filicic acid.[1]

Male fern is used in medicine to destroy or remove from the intestine tapeworms and the Ankylostomum duodenale, and most cases of poisoning have resulted from such use. As a rule, the drug passes through the bowel without being absorbed and without causing any effects except the death or expulsion of the worm, but sometimes, when large quantities are administered or when some unknown conditions favor the absorption of an unusually large amount of the active constituent, severe and even fatal symptoms have appeared. One of the conditions that have been thought to increase the absorption of the poison has been the administration of castor oil as a purgative after the administration of the male fern[2]; the castor oil is thought to dissolve the filicic acid, which is very toxic, and so increase its absorption. Whether or not this is the correct explanation, it seems to be true that in many cases of poisoning castor oil has been given.

The **symptoms** of poisoning by male fern have been due in part to its local irritating action upon the stomach and intestines and in part to the effects of stimulation and depression of the central nervous system. The chief symptoms have been vomiting, purging, acute abdominal pain, muscular weakness, confusion, and somnolence, with twitching of the muscles or slight convulsive movements, headache, fever, dyspnea, collapse, coma, and death. In some cases violent convulsions have occurred.[3] The urine has contained sugar, albumin, and blood. Icterus has often been observed, especially in the less severe cases; this has been attributed to duodenal catarrh, but may have been due to an increased destruction of the red blood-corpuscles in the liver.[4]

One of the most serious symptoms in poisoning by male fern has been blindness, either permanent or temporary. In 78 cases of poisoning collected by Sidler-Huguenin,[5] 18 of the patients became permanently blind in both eyes, while 15 became blind in one eye. Others became blind temporarily or there was permanent impairment of the vision. The lesion was atrophy of the optic nerve.

Fatal Dose and Period.—Children have died from 1 or 2 drams (3.7 or 7.4 c.c.) of the oleoresin of male fern. The fatal dose for adults has varied from 5 or 6 drams (18.5 or 22.2 c.c.) to an ounce and a half (4.5 c.c.),[6] but in one case it followed the administration of a little over 1 dram (3.7 c.c.).[7] Death has occurred in from six to twenty hours.[8]

Treatment.—In cases of poisoning fats and oils are contraindi-

[1] There is some doubt as to what the active constituents of male fern are. See Hausmann, *Arch. d. Pharm.*, 1899, vol. ccxxxvii., p. 544; also *Therap. Gaz.*, 1899, p. 179.

[2] Poulsson, *Arch. f. exper. Path. u. Pharm.*, vol. xxix., p. 24; Sidler-Huguenin, *Correspondenzbl. f. schweiz. Aerzte*, 1898, vol. xxviii., p. 513; a full bibliography is given with the latter paper.

[3] Eich, *Deutsch. med. Wochenschr.*, 1891, vol. xvii., p. 969.

[4] Grawitz, *Berlin. klin. Wochenschr.*, 1894, vol. xxxi., p. 1171.

[5] Sidler-Huguenin, *loc. cit.*

[6] *Lancet*, 1882, vol. ii., p. 630; *Boston Med. and Surg. Jour.*, 1882, vol. ii., p. 478.

[7] Paltauf, *Prag. med. Wochenschr.*, 1892, vol. xvii., p. 43.

[8] Eich, *loc. cit.*

cated; mucilaginous drinks and stimulants should be given after evacuation of the stomach and bowel.

Postmortem the stomach and intestines have been found congested and swollen and sometimes covered with small ecchymoses.

Detection.—There are no reliable tests for the active principles of male fern.[1]

SANTONIN.

Santonin (*Santoninum*, U. S. P., $C_{15}H_{18}O_3$), an anhydrid of santoninic acid, is contained in *Santonica* (U. S. P.), or Levant wormseed; the latter are the unexpanded flower-heads of *Artemisia pauciflora*.

Santonin is used almost exclusively as an anthelmintic to remove Ascaris lumbricoides from the intestine, and most cases of poisoning have occurred from its medicinal use. The greater part of the santonin passes through the intestine without being absorbed; under some little understood conditions, however, much of the santonin is absorbed and general symptoms of poisoning occur. The most common symptom of the general action of the poison is the production of xanthopsia, or "yellow sight,"[2]—objects that are brightly illuminated have a yellow tinge,—and there are often other disturbances of color vision. This effect rarely continues for twenty-four hours, although in one severe case of poisoning complete blindness lasted for nearly a week. Occasionally there are disturbances of the sense of taste, smell, and hearing. The symptoms[3] following the absorption of larger doses are due to changes in the central nervous system; they may begin within a few minutes.[4] There are headache, dizziness, trembling, unsteady gait, clonic convulsions,[5] and loss of consciousness. The convulsions may continue for several days, or the patient may early pass into a condition of stupor. These nervous symptoms are often preceded by abdominal pains and vomiting. Other symptoms observed have been perspiration, skin eruptions, edema of the skin, fever, and symptoms of irritation of the urinary passages—strangury, hematuria, and albuminuria. There is a tendency to a cumulative action on the part of the drug, as the latter is excreted slowly by the kidneys. The urine is yellow and may be diminished in quantity.

Fatal Dose and Fatal Period.—Nearly all cases of poisoning have occurred among children. At least 3 cases[6] are reported in which children from five to six years old died in from thirteen to fifteen hours after taking 6 grains (0.39 gm.) of santonin. A child of five died in thirty-five minutes after taking 6 grains (0.39 gm.) of santonin;[7] another child of the same age died in half an hour from an unknown amount.[8]

[1] Boehm, *Schmiedeberg's Archiv*, vol. xxxviii., p. 35.

[2] Rey, *Therap. Monatsh.*, 1889, vol. iii., p. 532; see Rose, *Virchow's Arch.*, vol. xvi., p. 233; vol. xviii., p. 15; vol. xix., p. 532; vol. xx., p. 245; vol. xxviii., p. 30.

[3] See, for example, Blinn, *Therap. Gaz.*, 1887, p. 497.

[4] Kilner, *St. Thomas's Hosp. Rep.*, n. s., 1880, vol. x., p. 246.

[5] A number of cases of santonin poisoning in which convulsions occurred are cited by Binz, *Arch. f. exper. Path. u. Pharmakol.*, 1877, vol. vi., p. 308.

[6] See Grimm, *Schweiz. Zeitschr. f. Med.*, etc., 1852, p. 492.

[7] Kilner, *loc. cit.*

[8] *Pharm. Jour. and Trans.*, 3 s., 1878, vol. viii., pt. ii., p. 996.

A child of three and a half years was severely poisoned for two days by $1\frac{1}{2}$ grains (0.1 gm.), but recovered; another child of four recovered after taking 4 grains (0.26 gm.). These and similar cases show that 2 or 3 grains (0.13 to 0.195 gm.) of santonin would be dangerous, and probably a fatal dose, for most children of from four to seven years of age. Adults have recovered from doses of from 8 to 15 grains (0.52 to 1 gm.). One-third of an ounce (10 gm.) of the crude drug caused the death of a child of ten years in two days.[1]

The **treatment** should consist in thorough evacuation of the stomach and bowels; ether, chloroform, or chloral hydrate may be necessary to control the convulsions. In case of collapse, warm baths with cold affusions are recommended. There are no characteristic **post-mortem appearances.**

Isolation.—Neumann has shown that santonin is partly decomposed in the body, giving rise to two substances, one of which appears in the urine and produces a red color with aqueous potassium hydroxid, while the other remains in the feces and loses its red color when treated with alkalis. Any undecomposed santonin, as well as these decomposition-products, may be recovered by either of the two following processes:

1. The material under examination is made into a thin paste with very dilute sodium hydroxid and digested at 30° C. with three times its volume of 96 per cent. alcohol for twenty-four hours. The liquid is filtered, the alcohol distilled off, and the aqueous alkaline fluid is shaken out with benzene. As long as the liquid is alkaline santonin will not be taken up by benzene, and various impurities may thus be removed. The santonin can finally be removed with chloroform or benzene after the aqueous fluid has been acidified with hydrochloric acid.

2. The material under examination is digested for several hours on the water-bath with milk of lime, and after filtration the alkaline fluid is treated with benzene as given above.[2]

Tests.—1. The material is placed on a watch-glass and exposed to the sunlight for a day or two.[3] On adding a drop of alcoholic solution of potassium hydroxid a characteristic red color is produced.

2. Neumann claims that the following modification of the Lindo[4] test will show the presence (0.0001 gm.) of santonin. A minute quantity of the suspected substance is warmed with a mixture of two volumes of concentrated sulphuric acid and one volume of water until a yellow color appears, when the material is allowed to cool and a few drops of 0.066 per cent. solution of ferric chlorid are added. On heating a second time a fine violet color appears.

3. When fused with potassium hydroxid, santonin imparts a red color to the mass.

[1] Linstow, *Vierteljahresschr. f. ger. Med.*, 1874, vol. xxi., p. 80.
[2] Neumann, *Jahresber. der Chem.*, 1884, p. 1645.
[3] Neumann, *loc. cit.*
[4] *Chemical News*, vol. xxxvi., p. 222.

VEGETABLE PURGATIVES.

A number of drugs of plant origin are used extensively in medicine as purgatives and occasionally give rise to poisoning. Most of the cases of poisoning are accidental, although some have been due to the use of the drugs for criminal purposes—for committing murder or for securing abortion. These substances differ considerably in their chemical properties, but their physiologic action is essentially the same in all cases. In small doses they simply hasten the normal movements of the intestines, while larger quantities cause all the symptoms of acute gastroenteritis. After poisonous doses there is violent purgation, accompanied by colic, griping pain, and tenderness in the abdomen; shock, collapse, and death may follow. Some of these drugs also cause local irritation when applied to the skin.

These purgatives may be divided into three classes, according to their chemical properties: (1) Purgative oils; (2) purgatives of the anthracene series; (3) the jalapin group.

PURGATIVE OILS.

Croton Oil.—Most cases of poisoning[1] with croton oil have been due to accident, as when preparations intended for external use have been swallowed,[2] or to the taking of too large medicinal doses. In rare cases it has been used for criminal purposes; thus strawberries[3] and cherries have been filled with croton oil and given for murderous purposes, or small doses have been given for a long period with the hope of causing death by setting up a chronic inflammation of the intestines. Death has also resulted from its administration in whisky as a practical joke.[4] A few cases of poisoning have resulted from the eating of the seeds of the plant.

Croton oil (*Oleum Tiglii*, U. S. P.) is a fixed oil expressed from the seeds of *Croton tiglium*. The pure oil itself is not irritant, but it is decomposed in the intestine into glycerin and crotonoleic acid. The acid is very irritant, and to this is due the purgative action of the drug. Most samples of the oil contain some free acid, and hence cause much irritation when applied to the skin or to mucous membranes.

Symptoms.—When taken internally, ordinary croton oil causes intense burning and irritation in the mouth and throat, excessive salivation, and vomiting. There are severe abdominal pain and profuse, often bloody, diarrhea, accompanied by much straining and tenesmus. The body is covered with cold perspiration, the pulse is small and irregular, the respiration slow and shallow; there are cyanosis and a fall of temperature. Finally there are delirium and collapse, and death from failure of the respiration. Occasionally death occurs without purging.[5] When applied to the skin, croton oil causes burning, redness, and vesica-

[1] Hirschheydt, *Dorp. Arb.*, 1890, vol. iv., p. 5.
[2] *Lancet*, 1870, vol. i., p. 553; Adam, *Edinburgh Med. Jour.*, 1856, vol. i., p. 932.
[3] Mayet and Hallet, *Ann. d'hyg. pub.*, 1871, vol. xxxv., p. 192.
[4] Ellis, *Amer. Jour. Med. Sci.*, 1874, vol. lxvii., p. 436. [5] Ellis, *loc. cit.*

tion; the vesicles may later contain pus. One-tenth of a milligram of croton oil applied to the tongue causes intense burning, continuing for hours.

Fatal Dose and Period.—Some specimens of oil seem to be more toxic than others; thus old specimens, which contain much free acid, are more toxic than fresh or purer samples, which contain but little free acid. This fact, as well as the circumstance that in most cases of poisoning some of the oil is returned in the vomit, makes it very difficult to determine the fatal dose. The condition of the stomach, whether atonic, filled with food, etc., also probably has some influence in determining the fatal dose.[1] One or two drops have caused severe poisoning, while 20 drops have caused death; a child of thirteen months died in six hours from $2\frac{1}{2}$ minims.[2] On the other hand, recovery has occurred after 1 dram and even after 1 ounce (30 c.c.) of the drug.[3] The medicinal dose is from $\frac{1}{3}$ to 1 minim. Death has occurred in four hours from $2\frac{1}{2}$ drams (9.25 c.c.); in another case, after a dose of less than a teaspoonful, death was delayed for three days. Death usually occurs within twelve hours. One-fourth of an old seed when chewed[4] has caused severe poisoning; a single seed is said to have caused death. The seeds contain, in addition to croton oil, poisonous toxalbumins. These are said to be especially abundant in fresh seeds.

The **treatment** consists in the thorough evacuation of the stomach by the stomach-tube or by the hypodermic injection of apomorphin. Demulcent drinks and morphin should be given to allay the pain. Cardiac and respiratory stimulants may be necessary.

Postmortem the mucous membrane of the alimentary tract is usually found reddened and swollen, and sometimes detached in places; hemorrhages may be found in the intestines. In some cases practically no changes have been found.

Detection.—The contents of the stomach and bowels, vomited matter, food, or other material to be tested should be finely subdivided, slightly acidified, if necessary, with tartaric acid, and repeatedly extracted with ether. The latter upon evaporation leaves as a residue any croton oil that may have been present in the material tested, together with other fatty substances soluble in ether. The residue may be tested for the presence of croton oil by rubbing a little of it on the inside of the arm and observing the eruption produced; and also by administering a small quantity to a cat, dog, or other lower animal and noting the gastro-intestinal effect. There are no conclusive chemical tests for croton oil in the small quantity in which it may usually be extracted from the contents of the stomach, and we are obliged, therefore, to rely upon physiologic experiments.

Castor oil (*Oleum Ricini*, U. S. P.) belongs to the purgative oils. It is similar, in both its chemical and physiologic properties, to croton

[1] Brydon, *Edinburgh Med. Jour.*, 1861, vol. vii., p. 134.
[2] *Med. Times and Gaz.*, 1870, vol. ii., p. 466.
[3] Bunting, *Boston Med. and Surg. Jour.*, 1868, vol. lxxviii., p. 294.
[4] Schulz, *Therap. Monatsh.*, 1889, vol. iii., p. 89.

oil, but its action is so much milder that it seldom, if ever, leads to severe poisoning.

The seeds from which castor oil is obtained contain the highly poisonous toxalbumin *ricin*, a body that has received a great deal of attention in the recent studies on immunity. Ricin does not pass into the castor oil, but severe and even fatal cases of poisoning may be caused by it when the castor-oil seeds are eaten. Stillmark[1] was able to collect 8 fatal cases up to 1889. The symptoms were vertigo, great weakness, and persistent vomiting; in most cases there was diarrhea, with rice-water, sometimes bloody, evacuations and tenderness and pain in the epigastric and umbilical regions. Later there were symptoms of severe collapse; small, frequent, barely perceptible pulse, cold perspiration, and death in from three to five or more days. In rare cases icterus, in others convulsions, were observed. In 16 cases of non-fatal poisoning observed by Edson[2] there was persistent vomiting but no catharsis; the patients were for the most part children under six. It is thought that only 3 or 4 seeds were eaten by each.

The severity of the symptoms seems to be somewhat independent of the number of seeds taken. Death followed, in six days, the eating of 2 seeds;[3] in another case death occurred in forty-six hours after eating 3 seeds; on the other hand, recovery followed the eating of 8 or 10 and of 25 or 30 seeds.[4]

Postmortem the mucous membrane of the stomach and intestine was found to be strongly injected, swollen, semidetached, and easily torn; there were numerous small hemorrhages.

THE ANTHRACENE PURGATIVES.

The purgatives, aloes, rhubarb, senna, frangula, and rhamnus (cascara sagrada), contain various derivatives of anthraquinon ($C_{14}H_8O_2$), and to these is due their purgative action. The only one of importance toxicologically is aloes.

Aloes.—Aloes is the inspissated juice of the leaves of *Aloe Perryi* or *Aloe vera*; the active principles are aloins, of which there are several varieties. Poisoning with aloes has resulted from the use of too large medicinal doses and from its use by the laity as an abortifacient. The chief symptoms are colic, abdominal pains, purgation, and tenesmus; the stools usually contain blood. Aloes has no specific action upon the uterus, but the increased flow of blood to the pelvic organs and the tenesmus caused by the drug may favor the expulsion of the fetus.

The **fatal dose** is placed at from $\frac{1}{3}$ to $\frac{2}{3}$ of an ounce (10 to 30 gm.).

Rhubarb (*Rheum*, U. S. P.) causes, in large doses, gastritis and enteritis, and, in certain susceptible individuals, skin eruptions.[5]

[1] Stillmark, *Kobert's Arbeiten d. pharmakol. Inst.*, Dorpat, 1889, vol. iii., p. 111.
[2] Edson, *Brooklyn Med. Jour.*, 1888, vol. i., p. 131.
[3] Meldrum, *Brit. Med. Jour.*, 1900, vol. i., p. 317.
[4] Hutchinson, *ibid.*, p. 1155.
[5] Goldenburg, *New York Med. Jour.*, 1889, vol. l., p. 652; Litten, *Therap. Monatsh.*, 1890, vol. iv., p. 606.

THE JALAPIN PURGATIVES.

A number of resinous purgative bodies may be grouped under the above heading. The most important of these are jalap, podophyllum, colocynth, scammony, elaterin, gamboge, and bryonia. The active principles of these drugs, so far as they are known, seem to be, in most cases, glucosids. All these substances are powerful purgatives, and some of them are contained in most of the purgative pills; the *Pilulæ Catharticæ Compositæ* (U. S. P.), for example, contain colocynth, scammony, jalap, gamboge, and also aloes, calomel, and cardamom. Death has resulted from the excessive use of proprietary pills containing various drugs belonging to this group;[1] in a case reported by Taylor such pills were used for the purpose of committing murder.

The action of all these substances is very similar; the symptoms following an overdose are vomiting, abdominal pain, violent purging, tenesmus, followed by great weakness, collapse, and death.

Jalap.—Jalap resin owes its activity largely to a glucosid, convolvulin; jalapin, another glucosid and the chief constituent of scammony, is contained in jalap in small amounts. The dose of the resin is from 2 to 5 grains (0.13 to 0.32 gm.).

Colocynth.—Colocynth (bitter apple), the fruit of *Citrullus colocynthis*, contains a glucosid, colocynthin. A number of deaths have followed large doses of colocynth; it has often been used as an abortifacient.[2] Poisoning has resulted from the inhalation of the powdered drug.[3]

The **symptoms** are the usual ones of gastro-intestinal irritation. There are also, at times, evidence of irritation of the kidneys and in fatal cases delirium, prostration, arrhythmic pulse, and collapse.

The **fatal dose** has varied greatly. Thus a woman died in twenty-four hours after taking a teaspoonful and a half of the powder, while in another case death followed less than 1 dram (3.9 gm.) of the powder. In a case reported by Tidy death did not occur until some time after forty hours after taking an unknown amount of the drug.[4] On the other hand, recovery has followed the taking of 2 and also of 3 drams (7.8 and 11.7 gm.). In the former case the woman was pregnant but abortion did not occur.[5]

Detection.—The isolation of colocynthin from the tissues is attended with some uncertainty. Tidy failed to find the poison in the case of a human being, but Dragendorff and Johannson[6] isolated it from the bodies of poisoned cats. The substance is colored first brown, then red, by Fröhde's reagent, while Mandelin's reagent (0.5 per cent. solution of ammonium vanadate in sulphuric acid) produces a transient blood-red color.

Podophyllum.—There occurs in the rhizome and roots of *Podophyl-*

[1] Paltauf, *Wien. med. Presse*, 1887, vol. xxviii., p. 579.
[2] *Pharm. Jour. and Trans.*, 3 s., 1878, vol. viii., pt. ii., p. 1035.
[3] See Jansen, *Therap. Monatsh.*, 1889, vol. iii., p. 39.
[4] Tidy, *Lancet*, 1868, vol. i., p. 158.
[5] Rolfe, *Boston Med. and Surg. Jour.*, vol. cxxvi., p. 494.
[6] Dragendorff, *Ermittelung von Giften*, Göttingen, 1895, p. 356.

lum peltatum (May-apple, a common North American plant, also called mandrake), a resinous body (*Resina Podophylli*, U. S. P.) the chief active principle of which seems to be a glucosid, podophyllotoxin.[1] The resin, commonly called podophyllin, is obtained by extracting the roots with alcohol and pouring the liquid into acidulated water, when the resin is precipitated. Fatal poisoning has resulted from the eating of the fruit of the May-apple, and the milk of cows feeding upon the plant has caused severe symptoms of poisoning in children. Severe conjunctivitis may occur in workmen who powder the roots or the resin.[2]

In a fatal case of poisoning by podophyllin reported by Dudley[3] the course was as follows: A woman took about 5 grains (0.32 gm.) of the resinoid podophyllin, instead of mandrake. Vomiting and purging began shortly after the drug was taken and continued for several hours. The extremities were cold; cold perspiration stood on the face; pulse 60, weak and thready; respirations sighing, and patient spoke with difficulty. The patient's condition rapidly improved, and she got up, but was soon compelled to lie down again. The primary symptoms of depression returned, her mind showed aberration, but there was no purging or vomiting. Twenty-four hours after the poison was taken patient was comatose; temperature 100° F., pulse 100, and respiration 38 to the minute. The urine was "smoky" and contained albumin and blood-cells. Patient died in a condition of coma about thirty-one hours after the drug was taken. The woman's husband took a similar dose and showed the same primary symptoms; these were followed by a depressed condition for two or three weeks and finally recovery. From these and similar cases the **fatal dose** may be placed at from five to ten grains (0.32 to 0.65 gm.), although recovery followed the latter dose when administered to a strong woman,[4] and a child twenty-two months old recovered from 4 grains (0.26 gm.).

Detection.—Both podophyllin and podophyllotoxin may be removed from an acid aqueous fluid, as in an examination for alkaloids, by shaking with chloroform. Both substances are colored dark yellowish green by Mandelin's reagent.

Gamboge (*Cambogia*, U. S. P.) is a gum-resin obtained from *Garcinia Hanburii*. It has occasionally caused death from its use as a purgative and as an abortifacient; about 1 dram (3.9 gm.) has proved fatal. Gamboge is used as a pigment, and children are said to have been poisoned by moistening their lips with brushes that had been used in the pigment.

Elaterium is a substance deposited by the juice of *Ecballium elaterium*, or squirting cucumber. Elaterium is of such uncertain strength that it is no longer recognized by the U. S. P., but the active principle, elaterin, a neutral body, is official under the name of *Elaterinum*. The latter is an extremely active purgative, the medicinal

[1] Podwyssotzki, *Arch. f. exp. Path. u. Pharm.*, 1881, vol. xiii., p. 29; Kursten, *Amer. Jour. Pharm.*, 1891, p. 485.
[2] Webster, *Medical Record*, vol. xii., p. 357.
[3] Dudley, *Medical Record*, 1890, vol. xxxvii., p. 409.
[4] Prentiss, *Phila. Med. Times*, 1882, vol. xii., p. 520.

dose of which is from $\frac{1}{60}$ to $\frac{1}{10}$ of a grain (0.001 to 0.007 gm.). A few deaths have resulted from elaterium.

The **fatal dose** is placed at 7 or 8 grains (0.455 or 0.52 gm.), although an ill and feeble lady of seventy seems to have died from the purging caused by $\frac{2}{5}$ of a grain (0.026 gm.).[1]

Detection.—The postmortem isolation of elaterin is not attended with certainty. The material under examination is extracted with hot alcohol, and the residue obtained after evaporation of the alcohol is boiled out with water, dried thoroughly, and extracted with ligroin, by which the poison is dissolved. Elaterin yields a carmin-red color on treatment with concentrated sulphuric acid that contains a trace of carbolic acid.

Treatment.—The treatment of cases of poisoning by the above purgatives is essentially the same as that mentioned under croton oil; the poison should be removed from the stomach by the stomach-tube or by emetics and demulcent drinks, and opiates should be administered. Great care should be taken with the diet, which should be as non-irritant as possible. Cardiac and respiratory stimulants may be necessary in conditions of collapse.

FIG. 67.—Poison ivy (*Rhus radicans*): *a*, Spray showing aerial rootlets and leaves; *b*, fruit.

Postmortem Appearances.—The only postmortem appearance commonly found is inflammation of the alimentary tract; sometimes there is ulceration of the entire gastro-intestinal canal. The peritoneum, liver, spleen, kidneys, and bladder have also been found inflamed.

RHUS TOXICODENDRON.

Several species of rhus, various members of which are popularly known as poison-ivy, poison-oak, swamp sumac, etc., give rise to frequent cases of poisoning. Speaking of poison-ivy, Chesnut[2] says: "No plant of the United States is more popularly recognized as harmful to man than this . . . . ; as its victims far outnumber those of all other plants combined, it has come to be regarded as *the* poisonous plant of America."

The active principle of these plants has been shown by Pfaff[3] to be

[1] *Amer. Jour. Pharm.*, 1868, p. 373.
[2] Chesnut, *Principal Poisonous Plants of the United States*, 1898, p. 36.
[3] Pfaff, *Jour. Exper. Med.*, 1897, vol. ii., p. 181.

a non-volatile oil, toxicodendrol; this oil occurs in all parts of the plant and is extremely active, $\frac{1}{1000}$ of a milligram ($\frac{1}{64000}$ of a grain) being sufficient to produce distinct effects when applied to the skin of some persons.

Poisoning usually results from direct contact with the plant, although this is not necessary; contact with articles that have touched or with people who have handled the plant is sufficient. The pollen, moreover, contains the oil, as do also the microscopic hairs[1] which are so abundant over the plant; these are responsible in many cases for "poisoning from a distance."

The most common form of rhus poisoning is that caused by the con-

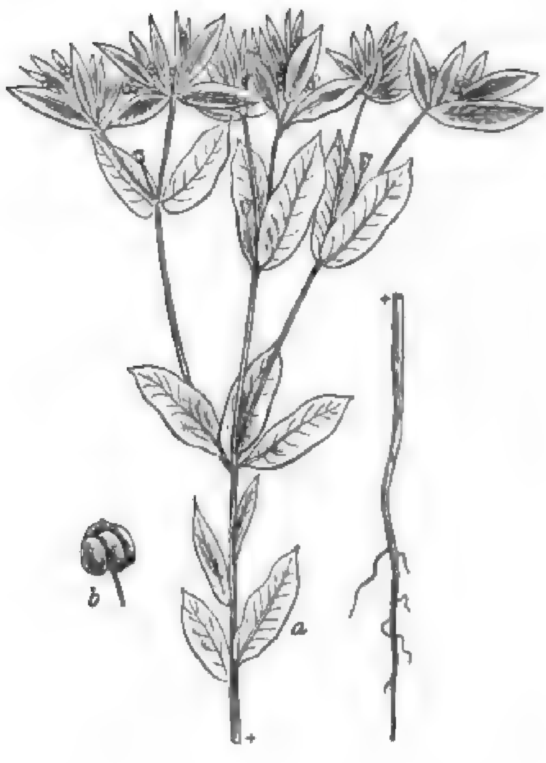


FIG. 68.—Snow on the mountain (*Euphorbia marginata*): *a*, Whole plant; *b*, seed capsule.

tact of the plant or of its juice with the skin, and consists in a severe inflammation and a vesicular eruption. There are violent itching, redness, swelling, vesication, and, finally, desquamation. In a case quoted by White,[2] deep ulcers formed which led to death in three weeks. If the poison reaches the face, the swelling may be so great as almost entirely to obliterate the features; the patient may be unable to open the eyes for several days. The period at which the first symptoms appear after

[1] Schwalbe, *Münch. med. Wochenschr.*, 1902, vol. xlix., p. 1616.
[2] *Dermatitis venenata*, p. 43.

exposure varies greatly; they usually begin within four or five days, but may appear in less than a day or be delayed for a week or even longer. The lesions of herpes zoster have been mistaken for those of ivy poisoning.

Poison ivy recently figured in a lawsuit in New York;[1] a judgment for $3500 was rendered against the directors of a cemetery in a case of poisoning due to poison ivy which had been allowed to grow within the grounds. The plaintiff was ill for eighteen months.

Of the numerous remedies employed in treating rhus poisoning, the

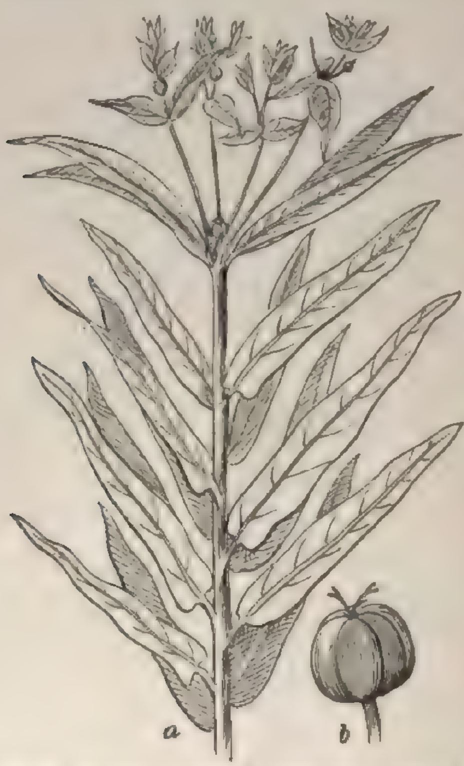


FIG. 69.—Caper spurge (*Euphorbia lathyris*): *a*, Upper half of plant; *b*, seed capsule.

best is probably an alcoholic solution of lead acetate; the alcohol brings the poison into solution, and the lead forms an insoluble compound with it. Vaselin and other ointments are contraindicated, for the toxicodendrol is soluble in fatty substances and their use simply aids in spreading the poison.

Moorman[2] reports a case of poisoning of children supposed to have resulted from the eating of the berries of poison ivy; there were symptoms of local irritation and also evidences of effects upon the nervous

[1] Trial Term of Supreme Court, Brooklyn, May 6, 1897; see Chesnut, *op. cit.*, p. 9.
[2] Moorman, *Amer. Jour. Med. Sci.*, 1866, p. 560.

system, as shown by drowsiness, stupor, and mild delirium. A case[1] is also reported in which an infusion of the root was taken instead of sassafras tea; there was a rash resembling that of measles, with intolerable itching, suffusion of the eyes, and pain in the throat and stomach. In experiments upon rabbits Pfaff found that the internal administration of toxicodendrol caused nephritis; perhaps the few deaths attributed to poison-ivy were due to this action upon the kidneys.

Detection.—To an alcoholic infusion of the material under examination is added an alcoholic solution of lead acetate, and the precipitated lead compound of toxicodendrol is suspended in water and decomposed with ammonium sulphid. After filtering off the lead sulphid the active principle is shaken out with ether.[2] As there are no chemical tests known by which toxicodendron may be identified, the residue obtained after evaporating the ether should be dissolved in olive oil and applied to the skin. The characteristic eruption thus produced may serve, under the most favorable conditions, for the detection of $\frac{1}{1000000}$ gram ($\frac{1}{64000}$ gr.) of the poison.[3]

A number of other plants, either native to the United States or introduced, are known to produce skin eruptions more or less similar to those caused by rhus;[4] among these may be mentioned various varieties of buttercups and crowfoot (Ranunculi), various species of Euphorbia, especially *Euphorbia corollata*,[5] which was formerly official, and *E. marginata*[6] or "snow on the mountains" (Fig. 68), and *E. lathyris* (Fig. 69), *Primula obconica*,[7] rue (*Ruta graveolens*), *Daphne mezereum*, and arnica. Several cases of poisoning have also been recently reported from handling various species of Cypripedium[8] (lady's-slipper, moccasin flower); the effects were very similar to those caused by rhus.

TANSY.

Tansy (*Tanacetum*, U. S. P.), the leaves and tops of *Tanacetum vulgare*, a common plant in the United States, contains a volatile oil. Tansy is used extensively by the laity as an abortifacient, usually without success, and a number of deaths have occurred from this practice; it is also used occasionally as an anthelmintic.

The **symptoms**[9] are similar to those caused by other volatile oils (see Oil of Turpentine, Savin, etc.); convulsions are common.

[1] Stokes, *Med. and Surg. Reporter*, 1867, vol. xvii., p. 373.

[2] Pfaff, *Jour. Exper. Med.*, vol. ii., p. 187.

[3] Pfaff, *ibid.*, vol. ii., p. 192.

[4] See White, *Dermatitis venenata*, 1887.

[5] See Olive, *Bull. Torrey Bot. Club*, 1895, vol. xxii., p. 393.

[6] See Jackson, *Med. Rec.*, 1897, vol. li., p. 636; Schenck, *Bot. Gaz.*, 1890, vol. xv., p. 277. Poisoning also results from the eating of honey derived from the flowers of *E. marginata*; the chief symptoms are vomiting and purging.

[7] See *Bull. Pharm.*, 1898, vol. xii., p. 80; Cooper, *Brit. Med. Jour.*, 1899, vol. i., p. 1543; Dreyers, *Münch. med. Wochenschr.*, 1902, vol. xlix., p. 574. A large number of cases are cited in the last article.

[8] See Chesnut, *Principal Poisonous Plants of the United States*, p. 19; *Northwestern Lancet*, 1898, vol. xviii., p. 382; *Bull. Torrey Bot. Club*, 1875, vol. vi., p. 115; Jessup, *Bot. Gaz.*, 1893, vol. xviii., p. 142.

[9] Belt, *Medical Record*, 1889, vol. xxxvi., p. 342: Jewett, *Boston Med. and Surg. Jour.*, 1880, vol. cii., p. 237; Dalton, *Amer. Jour. Med. Sci.*, 1852, vol. xxiii., p. 136.

It is impossible to state the **fatal dose** of tansy; 1 dram (3.7 c.c.) of the oil has caused death in an hour and a quarter,[1] while recovery has followed 2 fluidrams (7.4 c.c.)[2] and $\frac{1}{3}$[3] and, it is said, $\frac{1}{2}$ of an ounce (15 c.c.). Severe poisoning has resulted from $\frac{1}{2}$ fluidram (1.85 c.c.) and even from 15 or 20 drops. A patient died twenty-six hours after taking an infusion of perhaps $\frac{1}{2}$ ounce of the dry herb.[4]

OIL OF PENNYROYAL.

There are two preparations known as oil of pennyroyal—*Oleum Hedeomæ* (U. S. P.), derived from the common American pennyroyal (*Hedeoma pulegioides*), and the English oil of pennyroyal, derived from *Mentha Pulegium*. Both of these substances are used by the laity as emmenagogues; they have also a certain reputation as abortifacients,[5] and a few deaths are attributed to the use of the English oil. Thus a woman died after taking a tablespoonful of it,[6] the symptoms being those of gastro-enteritis.

PICROTOXIN.

Picrotoxin is a neutral bitter principle obtained from Cocculus indicus, which is the dried fruit of *Anamirta paniculata* (*Anamirta cocculus*, *Cocculus suberosus*, *Menispermum cocculus*), a climbing shrub of the East Indies; the fruit is also known as fish-berries, grains of paradise, and Levant nuts. The powdered berries have long been used as fish and bird poisons; they have also been used to a limited extent in medicine, chiefly as parasiticides and as a household remedy for vermin. At one time they were frequently added to beer to give it a stronger flavor when too little malt and hops had been used.

Most cases of poisoning from picrotoxin have been due to accident; in all the reported cases the berries or preparations of them have been used. The berries have been mistaken for wild cherries,[7] for cubebs, or for pepper, with fatal results. Death has resulted from the drinking of tinctures intended as remedies for vermin.[8] In a case cited by Taylor several men were poisoned by rum that had been impregnated with it. The application of a tincture to the head has caused death.[9] Poisoning is also said to have resulted from the eating of fish killed by Cocculus indicus.

Properties.—Cocculus indicus contains about 1 per cent. of picrotoxin. The latter (*Picrotoxinum*, U. S. P.) is a neutral body, acting, however, as a weak acid toward strong bases. The formula is probably $C_{45}H_{50}O_{19}$; on decomposition it yields two molecules of picrotoxinin

[1] Ely, *Amer. Jour. Med. Sci.*, 1852, vol. xxiv., p. 279.
[2] *Therap. Gaz.*, 1885, vol. ix., p. 342.
[3] Link, *New York Med. Jour.*, 1885, vol. xli., p. 365.
[4] *Phila. Med. Times*, 1881, vol. xi., p. 346.
[5] See Napier, *Brit. Med. Jour.*, 1890, vol. i., p. 661; Flynn, *ibid.*, 1893, vol. ii., p. 1270; Marshall, *ibid.*, 1890, vol. i., p. 542.
[6] Allen, *Lancet*, 1897, vol. i., p. 1022. [7] Shaw, *Med. News*, 1891, vol. lix., p. 38.
[8] Sozinskey, *ibid.*, 1883, vol. xliii., p. 485; Swift, *New York Med. Jour.*, 1897, vol. lxvi., p. 664. [9] Thompson, *Phila. Med. Examiner*, n. s., 1852, vol. viii., p. 227.

and one molecule of inactive picrotin ($C_{15}H_{18}O_7$). Picrotoxin is soluble in 240 parts of water at 15° C. (59° F.), and in 25 parts of hot water; it dissolves in 9 parts of cold alcohol, by which it is extracted from the seeds. It dissolves with difficulty in ether, chloroform, amyl-alcohol, and petroleum ether. It forms colorless, shiny, flexible crystals melting at from 192° to 200° C. (377.6° to 392° F.); it is odorless, but is intensely bitter.

Picrotoxin is a powerful stimulant to parts of the central nervous system. In the higher animals the action is almost limited to the medulla, and results in a series of powerful convulsions in which the clonic form predominates. There seems to be some involvement of the cerebral centers also, for in cases of poisoning unconsciousness is usually present; in many cases there is also a stimulation of the spinal cord, for tonic convulsions alternating with the clonic have been described, and there is also increased reflex excitability.

Symptoms.—A few minutes after the poison is swallowed there is felt a burning pain in the esophagus and stomach, followed shortly by salivation, nausea, vomiting, and diarrhea. There are weakness, confusion, dizziness, headache, drowsiness, cold, profuse perspiration, and unconsciousness; the face is pale, the pupils may be contracted or dilated, or these conditions alternate; the respiration is at first rapid and labored; later it is slow. Convulsions usually begin early (in twenty minutes,[1] for example); in Sozinskey's case they were as follows:[2] powerful general convulsions followed each other every five minutes or so, each lasting about two minutes. Between the convulsions there was perfect relaxation; hence they were very pronouncedly clonic. There was considerable opisthotonos. Each convulsion began with twitching of the muscles about the left corner of the mouth. As each attack came on there was more or less of an outcry, as in epilepsy, and some frothing was observable at the mouth.[3] In Thompson's case the pupils are described as being contracted during a convulsion and dilated between them; by touching the eyelid a spasm could be caused at pleasure, indicating an increased reflex excitability, as in strychnin poisoning. Consciousness is lost during the convulsions. The flexors are the muscles usually involved; sometimes, however, the jaws are firmly closed, as in strychnin poisoning. The pulse is sometimes weak; sometimes it is but little affected. Death results from respiratory failure.

If the patient survives the immediate effects, enteritis may result and lead to death several days later.

Fatal Dose and Period.—No cases of death from pure picrotoxin are reported; $\frac{1}{3}$ of a grain (0.022 gm.) has caused unpleasant symptoms, and from experiments on animals it is thought that 2 or 3 grains (0.13 or 0.195 gm.) would be a dangerous dose for man. Death is said to have resulted, however, from 36 grains (2.4 gm.) of the powdered berries; this would equal but about $\frac{1}{3}$ of a grain (0.022 gm.) of picrotoxin.

[1] See Wharton and Stillé, *Med. Jurisp.*, 1873, vol. ii., p. 598.
[2] See also Haynes, *Phila. Med. Times*, 1884, vol. xiv., p. 748.
[3] Sozinskey, *loc. cit.*

In several cases death occurred in thirty minutes. In the case reported by Swift death occurred in forty-five minutes; in a case reported by Rosenkrans,[1] within an hour; in Sozinskey's case it occurred in three hours. In Thompson's case, in which the drug was absorbed from the skin, death occurred in about six hours. In a case cited by Taylor death occurred from gastro-enteritis nineteen days after the poison was taken; in a similar case described by Mitchell[2] death occurred in twelve days.

Treatment.—The stomach should be evacuated by the stomach-tube or by emetics. Chloroform may be given during the convulsions. It is probable that moderate doses of chloral hydrate[3] would be of value, but this drug must be used with caution, for the picrotoxin kills in the same way as chloral does—viz., by paralyzing the respiratory center. Hot mustard-baths and other stimulants have been used in the late stages.

Postmortem Appearances.—In animals killed by picrotoxin there may be hyperemia and edema of the lungs and hyperemia of the meninges, and occasionally redness of the mucous membrane of the esophagus and stomach. No characteristic lesions have been found in man.[4]

Detection.—Picrotoxin may be extracted from an acid aqueous fluid with chloroform or amyl-alcohol, but the material obtained from animal substances in this manner is usually so impure that it will not respond to the tests, and various methods of subsequent purification have been suggested.[5] Palm[6] claims that all these modifications are defective, and gives the following process:

The material under examination is evaporated to dryness and extracted with acidified water. The picrotoxin is removed from the acid solution with ether, the ether evaporated, and the residue taken up again in water and decolorized with animal charcoal. Neutral lead acetate is now added, as long as a precipitate is formed, and after filtration the liquid is agitated with freshly precipitated lead hydroxid, which forms an insoluble compound with picrotoxin. The lead compound may be suspended in water, decomposed with sulphuretted hydrogen, and the picrotoxin extracted from the aqueous fluid with ether.

Tests.—1. A minute quantity of the suspected substance is evaporated to dryness with concentrated nitric acid, and the residue moistened with concentrated sulphuric acid. On the addition of an excess of potassium hydroxid, the presence of $\frac{1}{10000}$ gm. ($\frac{1}{650}$ gr.) of picrotoxin will be shown by the appearance of a brick-red color.[7]

2. Picrotoxin dissolves in concentrated sulphuric acid with a golden-yellow color that, upon the addition of a trace of potassium bichromate, changes to violet and then to brown. This test may be applied directly to the lead compound obtained in Palm's process.

[1] *Northwestern Med. and Surg. Jour.*, 1849, vol. i., p. 295.
[2] Mitchell, *Therapeutics*, 1850, p. 313.
[3] See Browne, *Brit. Med. Jour.*, 1875, vol. i., p. 540.
[4] *Cf.* Swift, *loc. cit.*
[5] Cheopinsky, *Jahresber. der Chemie*, 1884, p. 1644.
[6] *Zeitschr. f. anal. Chem.*, vol. xxii., p. 274; vol. xxiv., p. 556; vol. xxvii., p. 99.
[7] Langley, *ibid.*, vol. ii., p. 404; Cheopinsky, *loc. cit.*

3. Picrotoxin reduces Fehling's solution on warming with the production of red cuprous oxid.

CICUTA.

Several species of cicuta are very poisonous;[1] the one most carefully studied is *Cicuta virosa*, a common European plant. In this country *C. maculata*, which is very similar to, if indeed it is not identical with, *C. virosa*, is the most important. Other plants belonging to this genus are *C. vagans*, or Oregon water-hemlock, which is found in the far Northwest; *C. bulbifera*, which is found in the Great Lake States and also in the East, and *C. occidentalis*, found in the Rocky Mountains.

Boehm[2] and Pohl[3] have isolated from *C. virosa* a bitter, resinous, very poisonous principle, named by the former cicutoxin; its physiologic effects are very similar to those of picrotoxin. The plant also contains a non-poisonous volatile oil, cicuten. Apparently the American species have not been examined for cicutoxin, but from the symptoms produced by them, it is very probable that cicutoxin is the active agent. According to Glenk,[4] the fruit, but not the root, of *C. maculata*, contains an alkaloid similar to coniïn; it is not stated whether or not this alkaloid is poisonous. Strong[5] found 4.8 per cent. of ethereal oil, consisting largely of terpenes, in the fruit.

FIG. 70. Water hemlock (*Cicuta maculata*). Showing section of spindle-shaped root and lower stem, the leaves, flowers, and fruit, also fruit and cross-section of seed.

C. maculata (American water-hemlock; wild hemlock; spotted hemlock; spotted parsley; snakeweed; beaver poison; musquash root; muskrat weed; cowbane; children's bane; death of man).—According to Chesnut,[6] this is one of the most poisonous plants native to the

[1] At least some of the cases of poisoning attributed to *Sium latifolium* were very probably due to cicuta.

[2] *Arch. f. exp. Path. u. Pharm.*, vol. v., p. 284.

[3] *Ibid.*, vol. xxxiv., p. 265.

[4] *Amer. Jour. Pharm.*, 1891, p. 328.

[5] *Ibid.*, 1896, p. 236.

[6] Chesnut, *Principal Poisonous Plants of the United States*, U. S. Dept. of Agriculture, 1896, p. 40.

United States. All parts of the plant are poisonous, but the underground parts are especially dangerous; these have been mistaken, with fatal results, for horseradish, parsnips, artichokes, sweet cicely, angelica, and other edible roots. The plant is also very destructive to cattle.

The **symptoms**, which may not begin for some time after the plant is eaten,[1] are dizziness, violent, sometimes bloody, vomiting, cold skin, profuse perspiration, slow weak pulse, unconsciousness, and, finally, violent epileptiform convulsions, as in picrotoxin poisoning. The pupils are usually widely dilated; the jaw may be firmly closed.[2]

FIG. 71.—Oregon water hemlock (*Cicuta vagans*): *a*, Plant with leaves; *b*, root stock and horizontal roots; *b'*, section of root stock; *c*, terminal leaflets; *d*, flowering spray.

The **fatal dose** is not known; Chesnut says that a piece of the root of *C. vagans*, the size of a marble, is considered dangerous to man. A piece of the root of *C. maculata* the size of a filbert caused no unpleasant symptoms.[3] Of five persons poisoned by *C. occidentalis* in Montana in 1901, 4 died.

Death may occur in an hour.[4] In a case cited by Maisch[5] 3 children died within three hours after eating some of the root of *C. maculata*; in other cases life has been somewhat prolonged.

[1] In a non-fatal case reported by Folk (*Trans. So. Car. Med. Assoc.*, 1882, p. 69) there were apparently no symptoms for several hours.

[2] *Botanical Gaz.*, vol. x., p. 386.

[3] *Ibid.*, vol. xiv., p. 18.

[4] *Ibid.*, vol. xiii., p. 128; vol. xiv., p. 18.

[5] *Amer. Jour. Pharm.*, 1891, p. 322.

The **treatment** should be similar to that for picrotoxin poisoning—viz., evacuation of the stomach, the cautious use of chloral hydrate or of chloroform, and, in the later stages, of stimulants (rubbing the skin with flannel soaked in hot brandy, mustard baths, etc.).

No characteristic **postmortem changes** have been described.

Detection.—In the present state of our knowledge concerning the active principle of *C. maculata* the toxicologist would have to rely largely upon the microscopic appearances of any remnants of the plant found in the vomited matter. The coniïn-like odor is also of aid. European writers recommend that in cases of poisoning by *C. virosa* the contents of the stomach and intestines be extracted with ether and the residue be used for experiments upon animals. Cicutoxin, for which no chemical tests are known,[1] produces effects upon frogs almost identical with those caused by picrotoxin: among these effects are convulsions of medullary origin—*i. e.*, the convulsions disappear when the cord is separated from the medulla, but persist when the latter is separated from the forebrain. Such vital tests should be done only by an experienced pharmacologist.

FIG. 72.—Broad-leaf laurel (*Kalmia latifolia*): *a*, Flowering spray; *b*, vertical section of flower, showing peculiar attachment of stamens; *c*, fruiting capsules.

LAUREL.[2]

Many members of the heath family (Ericaceæ) are poisonous from the presence of andromedotoxin. The best known of these plants in the United States are *Kalmia latifolia* ("broad-leaf laurel," "mountain laurel," "mountain ivy," etc.), in which andromedotoxin was found by Plugge,[3] and *Kalmia angustifolia* ("narrow-leaf laurel," "lambkill," "dwarf laurel," etc.), in which the same principle was found by Lasché.[4] The former is found chiefly on rocky hillsides and mountain slopes up to from 3000 to 4000 feet from Connecticut to Eastern Ohio and along the Alleghanies to Georgia; the latter is abundant at low altitudes from Maine to New

[1] Dragendorff, *Ermittelung von Giften*, 1895, p. 297.
[2] See Chesnut, *Principal Poisonous Plants of the United States*, p. 44.
[3] *Arch. d. Pharm.*, 1889, p. 164 (cited in the *Amer. Jour. Pharm.*, 1889, vol. lxi., p. 361).
[4] *Pharm. Rundschau*, 1889, vol. vii., p. 208 (cited by Plugge, *Arch. d. Pharm.*, 1891, vol. ccxxix., p. 553.

Jersey, less abundant throughout the Great Lakes region, and south to Tennessee and South Carolina.

Little is known about andromedotoxin beyond the fact that it is free of nitrogen and that it is not a glucosid; the formula is $C_{31}H_{51}O_{10}$. It is contained in *Kalmia latifolia* to the extent of about 0.05 per cent. It is very poisonous, 1 to 2 milligrams ($\frac{1}{60}$ to $\frac{1}{30}$ of a grain) per kilogram animal being fatal to rabbits. The physiologic action of andromedotoxin is, in general, similar to that of aconitin.

But a few cases of laurel poisoning among human beings are reported. In some cases the poisoning resulted directly from eating the plant or from decoctions used in domestic medicine. The Indians are said to have sometimes committed suicide by drinking such decoctions. In other cases honey [1] derived from the nectar of the flowers has been held responsible. Shoemaker [2] reported 2 cases of poisoning from the eating of a pheasant in the craw of which laurel leaves were found. The symptoms were nausea, temporary blindness, pain in the head, dyspnea, pallid countenance, cold extremities, and feeble, slow pulse; both cases recovered. Chesnut has shown experimentally that the flesh of chickens fed upon laurel leaves is poisonous to cats. It is said that a decoction of laurel leaves is sometimes added to cheap whisky in order to increase its intoxicating effects. Many cattle and sheep are poisoned annually by eating the leaves.

Andromedotoxin [3] may be obtained in nearly pure form by dissolving the residue left after evaporation of an alcoholic extract in water, shaking this solution with ether, in which it is but slightly soluble, and then with chloroform. The andromedotoxin is extracted by the chloroform; by evaporating the chloroform, dissolving the residue in alcohol, and allowing the latter to evaporate slowly the andromedotoxin may be obtained in pure form. Andromedotoxin is more soluble in cold than in hot water; it is not precipitated by the alkaloidal reagents, and on heating with sulphuric and hydrochloric acids, a red color is produced.

LOCUST.

A few cases of poisoning are reported from the eating of parts of the common locust (*Robinia pseudacacia*); in some cases the leaves, in others the roots, but most frequently the bark, was the part responsible. Emery [4] described the poisoning of 32 boys from chewing the inner bark of the tree, which they had obtained from a yard where fence-posts had been stripped; the symptoms were vomiting, flushed face, dryness of the throat and mouth, and dilated pupils. In the more

[1] Coleman is said to have described in the *New Jersey Medical Reporter* for 1852, p. 46, the poisoning of 14 persons from eating wild honey supposed to have been collected from the flowers of *Kalmia angustifolia*; one patient died. The poisonous honey mentioned by Xenophon in the *Anabasis* was probably derived from *Rhododendron ponticum*, which contains andromedotoxin.

[2] *United States Dispensatory*, sixteenth ed., 1888, p. 1834.

[3] See Archangelsky, *Arch. f. exper. Path. u. Pharm.*, 1901, vol. xlvi., p. 318.

[4] Emery, *New York Med. Jour.*, 1887, vol. xlv., p. 92; see also *Phila. Med. Times*, 1880, vol. x., p. 387.

severe cases there were epigastric pains, extremely feeble, intermittent heart action, and stupor, from which the patients could be aroused with difficulty. The severity of the symptoms seemed to be somewhat independent of the quantity of bark chewed.

In a case cited by Lewin[1] the symptoms were similar, but there were also convulsive movements, as in belladonna poisoning, livid lips, and sunken eyes.

Cases of poisoning from eating locust leaves are reported from China[2]; a day or two after the leaves are eaten there are some fever and edema, first of the mouth, later of the entire skin. The edema of the lids is especially marked. In about a week there is desquamation of the entire skin, as in scarlet fever.

The toxicity of the leaves is said to be diminished by cooking.

According to the investigation of Power and Cambier,[3] the toxic principle of the bark is a toxalbumin. Kobert[4] found this substance to be similar to ricin; injected into the blood or under the skin, it caused fatal hemorrhages; the hemorrhages were especially severe in the intestine.

A glucosid, "robinin," has been obtained from the flowers.

[1] Lewin, *Lehrb. der Toxicol.*, 1897, p. 283; the case was described in the *Ann. de Therap.*, 1860, p. 64.

[2] Coltman, *Med. and Surg. Reporter*, 1889, vol. lxi., p. 236.

[3] *Pharm. Jahresber.*, 1890, p. 124.

[4] Kobert, *Lehrb. d. Intoxikationen*, 1893, p. 347.

GASEOUS POISONS.

OXYGEN alone supports human life. The inhalation of any other gas is followed by suffocation—that is, by suspended animation due to obstruction of blood aëration. Gases differ widely in their mode of action. Some, like nitrogen, merely exclude the oxygen of the air from the lungs, and the blood is then gradually exhausted of its oxygen. Life is restored in such cases when efforts to produce artificial respiration are not too long delayed and are sufficiently protracted. It may take an hour or more of persistent effort to revive a patient, and the rules laid down for the resuscitation of the drowned are applicable. Other gases, such as carbon monoxid, deprive the blood of its property of absorbing oxygen. They act directly upon the blood-corpuscles and are specific poisons. Such gases greatly retard or even prevent reoxygenation. Still other gases, like hydrogen sulphid, entering the system by absorption through the air-cells of the lungs, pass rapidly into the circulation and produce effects on special organs or on parts of the nervous system.

The only gases supposed to be purely negative in their action are nitrogen and hydrogen. To these the recently discovered argon may be added. There are no direct experiments on its inhalation. It is present in the air to the extent of nearly 1 per cent. without producing ill effects. Truly poisonous gases, even though present in much smaller quantity than this, seriously affect the organism. We are thus led to the assumption that argon is as passive in this as in all other respects. Since hydrogen when breathed with oxygen in atmospheric proportion has been found to produce narcotism, it should not, perhaps, be classed with nitrogen.[1]

Experiments made on rats confined in a space in which they could live in air without inconvenience for three hours showed the relative periods in which certain gases proved fatal.[2] Thus pure nitrous oxid killed in twenty-five seconds; pure hydrogen, in nine minutes; pure carbon dioxid, in eight seconds. Since experiments conducted by the Committee of the Medico-Chirurgical Society showed that the heart's action continued for eight minutes and twenty seconds under a complete deprivation of air, the above results indicate the truly poisonous action of vapors and gases, a conclusion substantiated by other facts.[3]

Although a large number of gases are poisonous, and although very many of them have at times produced death or serious effects, only the most important will be considered in detail in this section.

[1] Taylor, *Medical Jurisprudence*, eleventh Amer. ed., 1892, p. 439.
[2] Norris, *Brit. Med. Jour.*, 1873, vol. ii., p. 401. [3] Taylor, *op. cit.*, p. 493.

The following references to certain physical and chemical properties of the atmosphere have a bearing on the forensic as well as the hygienic side of this section :

Effect of Pressure on Respiration.
Bert, *Compt. Rend.*, 1872, vol. lxxiv., p. 617; vol. lxxv., pp. 29, 88, 491, 543.
Hoppe-Seyler, *Physiologische Chemie*, 1877, p. 7.
Effects of Ozone.
Hoppe-Seyler, *Physiologische Chemie*, p. 398.
Kobert, *Lehrbuch der Intoxikationen*, Stuttgart, 1893, p. 387.
Removal of Oxygen by Plants.
Phipson, "Analysis of the Air by a Mushroom," *Chem. News*, 1896, vol. lxxiv., p. 247.
Impurities in the Air.
C. F. Mabery, "An Examination of the Air of a Large Manufacturing City," *Jour. Amer. Chem. Soc.*, vol. xvii., p. 105.
Parkes, *A Manual of Practical Hygiene*, New York, 1884.
Woodman and Tidy, *Forensic Medicine and Toxicology*, Philadelphia, 1877, p. 856.
Newth, *Inorganic Chemistry*, London, 1895, pp. 231, 237, 648.
J. Th. Hermanns, "Ueber die vermeintliche Ausathmung gasfoermiger organischer Substanzen durch den Menschen," *Arch. f. Hygiene*, 1893, vol. i., p. 5.
Parkes, *op. cit.*, pp. 142 and 161.
Mason, *Water Supply*, New York, 1896, p. 50.
Annual Register, 1858, p. 278; letter of J. Z. Holwell, Esq., "Black Hole of Calcutta."
Hammarsten, *Lehrbuch der physiologischen Chemie*, 1895, p. 543.

CARBON DIOXID.[1]

(Chemical formula, $CO_2 = 44$. Synonyms, *Fixed Air; Choke-damp; Carbonic Acid; Carbonic Anhydrid.*)

Carbon dioxid is one of the commonest of all gases, being abundantly produced by a large number of natural and artificial processes, such as respiration, decay, alcoholic fermentation, combustion of coal and wood, lime-burning, etc. It is a heavy gas, having a specific gravity of 1.52 compared with air, and on account of its weight is rather slowly diffusible.[2] For these reasons it not infrequently is found in caves, old wells and cisterns, and other low and confined places, and has often been the cause of death of persons entering them.[3] Although it is only slowly diffusible, yet when once thoroughly mixed throughout the air, no separation of the gases subsequently takes place.

Action on Body.—Carbon dioxid acts *locally* on the point of application, or *remotely* on the brain and spinal cord. Applied to the skin, it produces a prickling or twitching, together with a feeling of warmth, which, if the stream is continued, is followed by anesthesia. In olden times local anesthesia was brought about by rubbing the skin at the place to be operated on with vinegar and powdered marble.[4] More profound symptoms arise when larger surfaces are affected, and bathing in water

[1] Consult also p. 226 *et seq.*, vol. i., in section on Death from Asphyxia.
[2] Wharton and Stillé, *Medical Jurisprudence*, vol. ii., p. 546.
[3] Briand and Chaudé, *Manuel Complet de Médecine Légale*, 6me ed., 1858, and 18me ed.
[4] Demarquay, *Compt. Rendus*, vol. lxi., p. 166.

charged with CO_2 causes a diminution of sensitiveness, reddening of the skin, warmth, prickling, and lowering of pulse-rate.

The gas disappears rapidly when injected subcutaneously without serious symptoms arising (Bernard). Others ascribe serious symptoms to the gas swallowed in aërated waters.[1]

Carbon dioxid renders arterial blood venous more rapidly than other gases. It acts on diluted blood as other weak acids. The hemoglobin is converted into acid hematin. Muscular tissue loses irritability and becomes rigid in gaseous mixtures containing much carbon dioxid. Ciliary movements are also arrested.

The brain and spinal cord are first stimulated, causing increase of blood pressure, peristaltic movement, and reduction in pulse-rate, followed by more intense respiration and less psychic excitability, and finally paralysis. Absorption takes place through all mucous membranes, the lungs, subcutaneous tissues, and also by the skin. Carbonates are changed to bi- or acid-carbonates in the blood or tissues.

The elimination is quantitative, and occurs by the lungs, urine, skin secretions, and intestinal discharges.

Death is ascribed in CO_2 poisoning, sufficient oxygen being supplied, to an excessive stimulation of the cerebrospinal system producing asphyxia, differing, however, from ordinary asphyxia, in which a loss of stimulation, especially of the heart, is due to the want of oxygen.[2, 3]

Symptoms.—These are either sudden or gradual, according to the degree of concentration of the gas breathed. Inhalation of the pure gas is followed by a spasm of the glottis, insensibility, and death from apnea unless the patient is speedily removed to a pure atmosphere. When the amount of CO_2 is smaller, there is a tendency toward giddiness, somnolence, and loss of muscular power. Profuse perspiration and nausea are common. The sudden loss of muscular power on entering a poisonous atmosphere is illustrated in the unfortunate accidents that are the most frequent cause of death by this poison. A workman enters a vat or other receptacle to clean it; a well, to dig it deeper—and is overcome. Another going to his rescue sacrifices his life, becoming powerless in the tainted air.

When the gas is in fatal proportion, there is a sensation of great weight in the head, of pressure in the temples, of singing in the ears, of a pungent sensation in the nose (similar to that given by drinking gaseous beverages), of giddiness, and of a loss of muscular power, and there is a strong desire to sleep.

If erect, the person falls to the ground as if struck; the body collapses, the head falling on the breast. The breathing, at first stertorous, becomes suspended. Convulsions may supervene, but not when a sufficiency of oxygen is present. The action of the heart, at first violent, ceases; sensibility is lost and coma follows. The body remains warm, the limbs flaccid, although occasionally rigid or convulsed. The countenance assumes a livid color (unless carbon monoxid is likewise present),

[1] L. Hermann, *Lehrbuch der exp. Toxikologie*, Berlin, 1874, p. 115.
[2] K. Kobert, *Lehrbuch der Intoxikationen*, Stuttgart, 1893, p. 689.
[3] L. Hermann, *op. cit.*, p. 121.

especially noticeable about the eyelids, lips, and throat. Sometimes, however, the face is pale and the features are placid. Death generally occurs without a struggle. Those resuscitated have complained of pain in the head or soreness of body lasting some days. Paralysis of the facial muscles has ensued. Persons may become gradually accustomed to the gas and continue to breathe an atmosphere that to any one entering would be unbearable.

Treatment.—Removal of the body as speedily as possible to fresh air, and inhalation, if possible, of oxygen; cold affusions, galvanism, artificial respiration, friction, and venesection are resorted to if necessary. That resuscitation may take place after long insensibility has been shown by experiments on animals and experience with human beings. The writer has observed pulsations of the heart in animals whose chests were opened after being many minutes in a concentrated atmosphere of carbon dioxid and when general lividity of the body indicated a deep-seated poisoning. The same animals serve over and over again for the demonstration of the poisonous effect of the air of the Grotto del Cane near Naples.

Postmortem appearances are those of death from asphyxia. Sometimes the face is livid and swollen and the features distorted, but not infrequently they are pale and placid; the position of the body indicates that the person has died without a struggle. The venous system and right heart are found filled with a dark-colored liquid blood, while the vessels of the lungs and brain are congested. There is nothing very characteristic in the general appearance, and death may be ascribed to apoplexy or to some other cause. Carbon dioxid being a narcotic poison, induces cerebral congestion and apoplexy.

Tests.—The air of a room contains too much carbon dioxid if a measured ounce of lime-water becomes turbid when shaken in a half-pint bottle of the air. This would be about 0.1 per cent. When the quantity reaches 0.5 per cent., most persons are attacked with languor and headache; while when the air contains the twelfth of its volume, suffocation is caused. The ordinary test of such confined air is the indication of the candle-flame, when any dimming of the same should warn against a person entering the space. The taper is extinguished when the proportion is above 12 or 15 per cent. Barium hydroxid is a better test than calcium hydroxid (lime-water). The air is shaken with or drawn through a quantity of this solution of known strength, and the loss of alkalinity determined by titration with dilute acid. Fifty c.c. of barium hydroxid solution are added to a bottle of 5 or 6 liters' capacity, which has been filled with the air of the room by the use of a flexible bellows pump. After thoroughly shaking the liquid with the air and after the lapse of half an hour, the turbid liquid is transferred to a stoppered separating funnel, the stem of which passes through a rubber stopper fitted to the neck of a bell-jar. One end of a piece of narrow flexible metallic tubing is inserted in the second perforation in the stopper, and the other end of the tube fits a small cork which closes the top of the tap-funnel. The bell-jar contains a small beaker, above

which rests a funnel provided with an asbestos plug, previously washed with baryta water. The bell-jar also contains sticks of moist potassium hydroxid to free the air of the jar of carbon dioxid. The barium hydroxid liquid is thus filtered in an atmosphere free of carbon dioxid. An aliquot portion of the filtered liquid is titrated with nitric or hydrochloric acid, phenolphtalein being used to indicate the point of neutralization.[1] When the amount of carbon dioxid is large, its quantity may be determined by observation with a eudiometer with the aid of potassium hydroxid solution. The dried air may also be drawn through a weighed vessel containing potassium hydroxid solution, when an increase in weight will show the amount of carbon dioxid in the volume of air submitted to analysis.

When the air of a well, vat, or similar receptacle is to be tested, a sample for examination may be obtained by lowering into it a bottle containing fine sand and inverting by means of a cord attached to the bottom ; when the sand running out is replaced by the air, the bottle is raised, mouth up.

CARBON MONOXID.[2]

(Chemical formula, CO = 28. Synonym, *Carbonic Oxid.*)

Carbon burns directly to carbon monoxid at 1000° C.[3] It is produced at the electrodes or from the charges of electric furnaces. It is also thus produced in blast furnaces. In electric furnaces having limestone linings the carbon dioxid is reduced to carbon monoxid by the heated electrodes. The gas escapes unburned, producing characteristic symptoms.[4]

The commonest source of carbon monoxid is its production in coal-fires of grates, stoves, and furnaces (domestic or industrial). The blue flame at the surface of the fire shows its presence. Much escapes unburned. In furnaces with forced draft the blue flame is often seen at the top of the chimney, and in iron blast-furnaces the gas is conducted and burned in the superheaters of the air-supply. It is, therefore, a constant constituent of the products of combustion of carbonaceous materials, and while the poisonous action of charcoal, coke, or coal vapors is in part due to carbon dioxid, it is mainly due to this more poisonous gas. Quantities of this accumulate above the fire in stoves and hot-air furnaces. So much, indeed, that on opening the door the inrush of air will form an explosive mixture that, igniting, may produce serious accidents. The common construction of dampers is that they shall be loose enough to allow a constant draft to the chimney. Notwithstanding this it frequently happens that a down draft fills the room with the products of combustion to a poisonous extent. A large proportion of the accidents that happen yearly is due to the leaving-off

[1] Williams, *Commemoration Volume, University College*, Sheffield, 1897, p. 132.
[2] Consult also p. 237 *et seq.*, vol. i., in section on Death from Asphyxia.
[3] *Bloxam's Chemistry, Inorganic and Organic*, eighth edition, Philadelphia, 1895, p. 95.
[4] Moissan, *Le Four Electrique*, Paris, 1897, p. 15.

of the stove-lids. In Europe the charcoal brazier or the portable stove is the chief source of trouble.

Carbon monoxid has been detected by the writer in the gases escaping from fires made with briquettes of "smokeless fuel." As the result of several accidents, notably one which happened to Dr. Motet, an ordinance was passed in Paris prohibiting the heating of cabs by the use of such briquettes. The law is, however, a dead letter. Nevertheless the record of the poisoning is undoubted. The briquettes were burned in foot-warmers and rendered the air of the closed vehicle toxic from the presence of carbon monoxid. The symptoms produced were severe.[1]

Carbon monoxid is formed when gas-flames impinge on a besooted surface. It is also produced in lamps turned low; in mines by the explosion of gun-powder and other explosives, and is thus frequently encountered in the air of mines after blasting. Many casualties have occurred from the production of this gas by the slow combustion of beams, flooring, and other woodwork in the neighborhood of closed rooms, and it has been a matter of considerable importance to determine whether death was due to the influence of this gas previous to the charring of the body, or whether the gas found in the blood was due to post-mortem absorption, the result of the carbonizing of the corpse. Several instances of the latter are given by von Hofmann and Brouardel.[2]

It was thought at one time that carbon monoxid passed through heated iron. An important investigation was reported to the French Academy. Subsequent investigation, however, has indicated that such transpiration does not take place. The presence of any carbon monoxid in the atmosphere is due either to the faulty construction of the furnace or to the action of the heated plates on the organic dust with which they are generally covered.[3] Notwithstanding all attempts to render furnaces gas-tight, their lack of this is seen in the rapid tarnishing of silverware as soon as the furnace is lighted; by the occasional escape of furnace gas, detectable by its odor; or of smoke, which pervades the house. The general malaise and the headaches during the winter months are undoubtedly due in many instances to the presence of this gas.

Enormous quantities of carbon monoxid are daily produced in the manufacture of illuminating gas, producer gas, and water gas. Of the 40,800,000 cubic feet of street gas daily distributed in New York city through the 1491 miles of mains, over 10 per cent. is carbon monoxid.[4] Water gas contains about 30 per cent. of carbon monoxid, producer gas slightly less; blast-furnace gas about the same as producer gas, while lime-kiln gases, waste gases from ammonia soda processes, and gases left in the track of explosions in coal-pits contain from 1.25 to 2.5 per

[1] Brouardel, *Les Asphyxies*, Paris, 1896, p. 29.

[2] Kobert, *Lehrbuch der Intoxikationen*, Stuttgart, 1893, p. 525

[3] Remsen, "Carbonic Oxid as a Source of Danger to Health in Apartments heated by Cast-iron Furnaces or Stoves," *National Board of Health Bulletin*, Washington 1881, vol. ii., No. 52, p. 857; J. Gottschalk, *Ueber die Nachweisbarkeit des Kohlenoxydes in sehr kleinen Mengen und einige Bemerkungen zu der sogenannten Luftheizungsfrage.*

[4] *Annual Report of the State Board of Health*, Department of the City of New York, 1895, p. 206.

cent.[1] Wood gas made by the Wilkinson process was found to contain 33.75 per cent., while the commercial gas served to the consumer and made by mixing of gases from wood, coal, and naphtha contained 11.25 per cent.[2]

Carbon monoxid is a colorless gas of very faint odor and is nearly insoluble in water. It is lighter than air, having a specific gravity of 0.967. It has been liquefied and solidified. It combines with metals such as nickel and iron to form colorless liquids called carbonyls. Heat decomposes these with deposition of the metal and evolution of the gas.

It does not support combustion, but forms an explosive mixture with one-half its volume of oxygen or two and one-half volumes of air. It is highly poisonous.

Action.—Carbon monoxid may be freely respired, its presence in the air not being manifested either by irritation to the air-passages or by its affecting the sense of smell; but the moment it comes in contact with the blood, by diffusion, it unites with the red pigment of the blood-corpuscles forming a definite compound, carbon monoxid hemoglobin, exactly replacing the oxygen volume for volume. This compound with the red pigment is singularly stable, the affinity of carbon monoxid for the hemoglobin being 200 times stronger than that of oxygen for the same substance; as a consequence, in carbon monoxid poisoning the blood cannot readily become arterial. The blood-corpuscles are not changed in form, though those found in the liver are, after a time, somewhat modified. The gas causes the blood to assume a color varying from violet to cherry red, and both blood and foam are readily distinguished from normal arterial blood. Blood containing carbon monoxid hemoglobin may be deprived of the gas by submitting the blood to diminished pressure or by the passing of oxygen or air through it for a considerable length of time.

From observations on human beings some claim that this change may take place in a couple of hours, while others state in from four to six. It is claimed that the carbon monoxid may be in part changed by oxidation into carbon dioxid within the body (Gruber). Others, among them Gaglio, claim that this oxidation does not take place, but that the carbon monoxid is voided, quantitatively, unchanged.[3] Carbon monoxid when injected into the abdomen or swallowed, dissolved in water, also produces poisonous effects as if inspired. The blood is, however, never fully saturated, and some of the gas may diffuse from the corpuscles into all the tissues; indeed, it has been shown by Fehling, from the mother to the fetus. This raises the question of whether a corpse may not absorb the gas from the atmosphere and thus present indications of carbon monoxid poisoning when death has been due to other causes.

[1] Douglas Herman, "Notes on Poisoning by Carbonic Oxide," *Journal of the Society of Chemical Industry*, 1896, vol. xv., p. 857.

[2] C. A. Doremus, "Wilkinson's Process of the Manufacture of Illuminating Gas from Wood," *Jour. Amer. Chem. Soc.*, 1880, vol. ii., p. 419.

[3] Concerning the elimination of carbon monoxid consult L. de Saint-Martin, "Recherches sur le mode d'élimination de l'oxyde de carbone," *Comptes Rendus*, 1891, vol. cxii., p. 1232; 1892, vol. cxv., p. 825.

The question as to whether carbon monoxid has any direct action upon the nervous system has been the subject of much dispute. The inhalation of oxygen with at least 20 per cent. of carbon monoxid seems to affect the nervous system, since violent disturbances of the system with cramps and total paralysis of the limbs appear within the first minute of inhalation, while the blood could certainly not have become sufficiently saturated with the gas by that time to produce such symptoms. Kobert, Geppert, and others strongly incline to the belief of an action upon the nervous system, both of the peripheral nerves and of the ganglion-cells of the brain, from the symptoms produced and the pathologic appearances, and they extend the poisonous action of the gas to the production of a degeneration of the muscles and glands.[1]

Lethal Dose.—Since the poisonous action of this gas was noticed in 1802 by Guyton de Morveau and submitted to personal experiment by Sir Humphry Davy, a large number of experiments have been made on men and animals and observations recorded from the results of accidents. It would appear from these that about 0.8 gram (about 12 grains) of carbon monoxid is fatal to a man of 70 kilos (154 pounds), 11.5 milligrams per kilo being fatal to rabbits. From this it would appear to be less poisonous than prussic acid. Nevertheless, extremely small portions when breathed produce unmistakable symptoms of poisoning. According to Gruber, 0.02 per cent. is the limit of toxicity, while at 0.05 per cent. symptoms were clearly observable.[2]

It is especially noticeable that all animals do not behave alike. In some, as birds and chickens, convulsions or cramps are caused, while this effect is not noticed in mice or rabbits. Gruber found rabbits behaved abnormally in an atmosphere containing between 0.07 and 0.08 per cent. As soon as the quantity reaches 0.1 per cent., the poisonous effect is produced, while with 1 per cent. the toxic action is rapid. Gruber breathed air containing 0.021 to 0.024 per cent. of carbon monoxid for over three hours without experiencing any unpleasant sensations. He states that the blood of the entire body is capable of containing one liter of oxygen or one liter of carbon monoxid. The quantity of carbon monoxid breathed by him amounted *in toto* to 300 c.c., but being distributed over a considerable length of time, did not produce poisonous effects. Frogs placed by him in an atmosphere of pure carbon monoxid lived over ten hours.

The rapidity of the action has been established by many observations. Birds die instantly in an atmosphere containing 5 per cent., while dogs, rabbits, and other animals are killed in times varying from a minute or so to half an hour, according to the amount of this gas in the atmosphere.[3]

The speed with which carbon monoxid acts is illustrated by a case

[1] Kobert, *Lehrbuch, op. cit.*, p. 527; also Kobert, *Practical Toxicology*, English translation, L. H. Friedburg, New York, 1897.

[2] Max Gruber, "Ueber den Nachweis und die Giftigkeit des Kohlenoxyds, und sein Vorkommen in Wohnraeumen," *Arch. f. Hygiene*, 1883, vol. i. p. 145.

[3] Woodman and Tidy, *Forensic Medicine and Toxicology*, Philadelphia, 1877, p. 485.

given by Sonnenschein of a chemist who, by a single breath of an atmosphere laden with this gas, fell backward as if struck by lightning. He recovered after a quarter of an hour's serious symptoms through timely aid.[1]

Carbon monoxid has, through experiments on water-gas, been shown to combine directly with nickel and iron, producing carbonyls, as nickel carbonyl, $Ni(CO)_4$ (Mond). This substance has been found to be poisonous, with symptoms similar to carbon monoxid poisoning. Phosgene gas, carbonyl chlorid ($COCl_2$), carbonyl sulphid (COS), and several condensation products have all been found to be more or less poisonous.

Symptoms.—Unless accidental, pure carbon monoxid poisoning is rarely met with in the human subject, but since many of the gaseous mixtures contain a high proportion of the gas, the other gases being of a less toxic character, we may fairly assume the symptoms in these cases to be due to carbon monoxid. A large number of experiments on animals made to breathe carbon monoxid, diluted with either oxygen or air, have resulted in showing that the blood pressure is at first considerably increased as the result of the stimulation of the vasomotor center. A benumbing of this center is then shown by the decrease of the pressure and a distention of the blood-vessels. Apoplexy may result from the first stage. More or less extended patches of bright color, especially on the anterior portion of the body, make their appearance and are distinctly different from the violet-red patches that appear postmortem, especially on the dependent portions of cadavers. The pulse becomes slower when the blood pressure rises, and a violent beating of the heart is experienced. Subsequently the pulse becomes frequent, but small. Breathing is deep and difficult. Respiration ceases when the respiratory center is paralyzed, but the stage of stimulation is protracted. The muscular system is very generally affected, in which case the extremities may fail to perform their functions for many days, or special muscles or muscle groups become paralyzed and afterward degenerate. Sensation to pain may be absent or remain suspended for a long time. The sensations are headache, throbbing of the temples, singing in the ears, faintness, dizziness, and vomiting. The face becomes red, and there is loss of memory, vertigo, fainting, anesthesia, and loss of all spontaneous power of movement, together with tonic and clonic spasms. The heart's action is at first violent in the stupor, then weak, slow, and arrested. The bodily temperature is lowered. Involuntary discharge of feces, urine, and seminal fluid is not infrequent.

The recovery is sometimes rapid. Men working around gas-mains, in ditches, will be affected, but when taken into the air or given a drink of whisky or other stimulant speedily recover and return to work. As a rule, however, there is a slow return to consciousness, with more or less prolonged headache, nausea, and weakness. Symptoms may continue for several days. Where the gas has been inhaled for a considerable time, the red patches on the skin will remain for quite a while. The paralysis and anesthesia begin in the lower extremities and rise to

[1] F. L. Sonnenschein, *Handbuch der ger. Chemie*, Berlin, 1869, p. 288.

the trunk. The loss of power and of sensibility is frequently shown by the severe burns received by a person falling on a gas- or other stove or brazier. Loss of consciousness is often sudden. At other times there is a slowly increasing drowsiness. There is a great similarity in the symptoms to those of drunkenness. Recovery may in some cases follow a protracted sojourn in a not too poisonous atmosphere, while others, after an hour or two's inhalation, cannot be brought to life. While most have no remembrance of the symptoms, many claim to have suffered greatly. Death follows from paralysis of the respiratory apparatus. When the gas itself does not kill, apoplexy or softening of the brain may follow. According to Becker and Schwerin, the sequelæ divide themselves into four groups: (1) Primary gangrene with blisters and decubitus. (2) Primary hemorrhages, as of the lungs, apoplexy, and the like. (3) A persistent distention of the capillaries and other vessels in which the symptoms are shown in the skin, red nose, red spots not unlike those caused by frost-bite. (4) A deep-seated disturbance of the regeneration of all organs, especially of the vascular walls and of the ganglion cells of the nervous system, evidenced by secondary hemorrhages, idiocy, imbecility, chorea, ascending paralysis, etc. Indeed, the results of this variety of poisoning are manifold, reminding one of diseases of the brain, spinal cord, lungs, kidneys, liver, or skin. Diagnosis is difficult.

Chronic poisoning by carbon monoxid has received the attention of many observers in recent years. There is very good evidence of this form. Accumulated cases show that it is the result of being in a constantly contaminated atmosphere. The symptoms are described as an alteration in the digestion, diminished vigor, gray color of the skin, coated tongue, loss of memory, diminution of the psychic powers, and occasional convulsions. The pathologic findings of autopsies have shown, in some cases, fatty degeneration, in others, pernicious anemia.

Gruber[1] has shown, along with others,[2] that carbon monoxid is not a cumulative poison, but that when inhaled in very small quantities, it disappears from the blood. In cases of chronic poisoning the blood does not serve for diagnosis. This is not the case, however, in acute poisoning, where a timely examination of the blood taken from the patient as soon as possible will indicate the poisonous effect of carbon monoxid and lead to the adoption of proper methods of resuscitation. As a further aid to diagnosis, the comatose condition, low temperature, absence of odor of alcohol, loud snoring, and bright-red appearance of the face would lead one to suspect gas-poisoning, especially if the body were found in a closed room or in a confined space with indications of gas-poisoning more or less apparent. As a further diagnosis the urine is found to contain a reducing substance, generally glycuronic acid, and in many cases there is albuminuria. Red patches or spots upon the surface of the skin are characteristic.

[1] Gruber, *op. cit.*

[2] "A Study of the Poisonous Effects of Coal- and Water-gas," W. T. Sedgwick and W. R. Nichols, *Sixth Annual Report of the State Board of Health and Charity of Massachusetts*, Boston, 1885.

Treatment.—This consists in taking the person at once out of the poisonous atmosphere into fresh air, and in inducing artificial respiration as rapidly as possible. The giving of oxygen under slight pressure, enough to distend the cheeks, and then compressing the lungs, is considered one of the most, if not the most, efficient method of restoring the person to life. Unfortunately, the cost of this gas is apt to restrain it from being used in sufficient quantity. If the body possesses an abnormally low temperature, artificial warmth is needed. Cold affusions to the head, injection of iced water, friction, mustard baths, faradization of the phrenici, or stimulation with ammonia are simple expedients. Hydrogen dioxid and ozone, though recommended, have not been found efficient as antidotes. The recovery in a recent case [1] of gas-poisoning is, however, attributed to the prolonged use of the former, it being given in doses of two to three ounces, diluted with water, both by the stomach and as an enema. Black coffee and alcoholic stimulants may be given if the person can swallow. Transfusion of the blood has been recommended, but has not proved very satisfactory. Injection of alkaline salt solution directly into the veins, or, according to more recent practice, into the rectum, the body being placed in the position of auto-transfusion, recommended by Jersey, has been often resorted to. Venesection of from 6 to 12 ounces precedes the introduction of the salt solution.[2] The frequent occurrence of accidents known as "gassing" has led to having compressed oxygen at hand in works, mines, and other establishments. Experience has shown that a single person is, as a rule, not able to rescue any one overcome.[3]

Either the "pneumatophore" (Fig. 73) of Wallach Bros. or the "safety hood" (Fig. 74) of Ziemer and Co. can be used in conjunction with the small portable oxygen bottle, from which the oxygen under high pressure is released into the rubber bag or into the hood, to be breathed therefrom under low pressure. The hood can likewise be supplied with air by means of a long armored india-rubber tube attached at a distance to a double-acting air-pump.

The consensus of opinion at this writing seems to be in favor of the use of oxygen in carbon monoxid poisoning, and especially of having the remedy at hand in all works where operatives are exposed to noxious atmospheres. It is reported that no case of suffocation, though cases have been numerous, has proved fatal since the introduction of the oxygen bottle [4] (Fig. 75).

The following case is cited:[5] Four men engaged in painting the interior of a boiler with an anticorrosive liquid were rendered insensible by vapors evolved before they could give warning to those outside.

[1] C. J. Lind, *Northwestern Lancet*, Feb. 15, 1902, p. 70.

[2] W. T. Bull, *Med. Record*, New York, January 5, 1884.

[3] "The Use of Oxygen in Rescue Work in Collieries," *American Gaslight Journal* (from the *Colliery Guardian*), 1897, vol. lxvii., p. 108; George Davis, *Handbook of Chemical Engineering*, 1902.

[4] Douglas Herman, "Notes on Poisoning by Carbonic Oxid," *Jour. Soc. Chem. Industry*, vol. xv., pp. 854–858.

[5] *American Gaslight Journal*, December, 1896.

The head boiler-man, entering the boiler to ascertain the cause of the silence, was also overcome before he could withdraw, and a like fate overtook another workman, so that there were six men lying unconscious in the boiler. After a lapse of several hours the works' engineer was summoned. He immediately emptied two oxygen bottles, each containing 220 gallons, into the boiler, whereupon the heavy breathing of the sufferers became easier, and soon after four of the men crawled out unaided. The other two lay between the wall of the boiler and the sliding flue, and to them a supply of 220 gallons of oxygen was delivered through a hose, with the result that within a short time they, too, were able to make their way out; and although they had been exposed to the poisonous atmosphere for some hours, they fully recovered within a few days.

In another case a man was overcome by the backdraft from a blast furnace in the course of charging. Pure oxygen was introduced

Fig. 73.—Pneumatophore.

Fig. 74.—Safety hood respirator.

through the nose. He recovered consciousness and walked home. No details were printed.

In still another case oxygen restored consciousness, though bleeding, electricity, injections, friction, and attempts at artificial respiration had failed.[1]

Blake[2] reports that three out of four men overcome by coal-gas were restored by oxygen. The inhalation of oxygen prevents the headache subsequent to poisoning.

Suitable helmets are now obtainable for the use of firemen who are forced to enter buildings filled with noxious fumes. Ambulances and fire-engines should, therefore, be equipped with such rescue apparatus.

[1] Douglas Herman, "On Poisoning by Gas: its Prevention and Cure," *Jour. Soc. Chem. Ind.*, vol. xv., p. 247.

[2] Blake, *Boston Med. and Surg. Jour.*, 1872, vol. lxxxvii., p. 149.

Postmortem Appearances.—These consist in the cherry-red color of the body (Plate 11) and internal organs, the postmortem patches, and the spectrum of the blood. There is a degeneration of the heart, liver, kidneys; ruptures of the vessels of the brain; blisters of the skin, and anemia. The urine contains sugar, glycuronic acid, and lactic acid. There is no specific odor of the cavities of the body or of the blood, though in many reports of autopsies the statement is made that the witnesses were more or less affected with symptoms of gas-poisoning. In chronic cases there are signs of softening of the brain and degeneration of other organs.[1,2]

Stevenson[3] reports a case where the condition of the viscera was phenomenal, the rosy hue being visible after seventeen months. This is also the case with blood, which a great many observers say retains its color for several years, observations which the writer has confirmed by his own experiments.

FIG. 75.—Oxygen bottle and fittings: *c*, Oxygen bottle; *b*, stand; *s*, main valve; *k*, key; *u*, union; *r*, regulating valve; *t*, key; *n*, nipple for hose.

Otto[4] states that Landois was able to detect carbon monoxid in the body of a woman eighteen months after death. The blood retained its red color notwithstanding the fact that the body had undergone extreme putrefaction. Otto himself preserved the blood for two years by keeping it in a closed vessel.

Tests.—Carbon monoxid may be detected in the air through the absorbent power of a solution of cuprous chlorid in an excess of hydrochloric acid or an excess of ammonia. In either liquid it is completely absorbed. When large quantities exist, the diminution of the volume of the air passed through this reagent may be observed, but generally large volumes of the air have to be drawn through the solution, and the presence of the gas determined either by heating the solution to boiling, which causes its expulsion, or by dropping the same in a bright-red solution of palladous chlorid, whereby a black precipitate of metallic palladium is produced ($PdCl_2 + CO + H_2O = CO_2 + 2HCl + Pd$). The writer has been able to detect the carbon monoxid generated by the burning of a smokeless fuel by the former method, absorbing the gas in cuprous chlorid solution, and afterward expelling it. Tests were applied, and the gas found to burn with a pale-blue flame. Care must be taken in using the palladium solution to exclude the presence of hydrogen sulphid, ammonium sulphid,

[1] Lesser, *Atlas der ger. Med.*, Berlin, 1883, vol. i., p. 141.
[2] Hofmann, *Atlas der ger. Med.*, Munich, 1898, plate 51.
[3] *Guy's Hospital Reports*, 1890.
[4] Otto, *Anleitung zur Ausmittelung der Gifte*, seventh ed., Braunschweig, 1896, p. 262.

Carbon Monoxid Poisoning (Von Hofmann).

A woman was found dead before her bed, lying with her face downward. In her small room there was a basin containing half-consumed charcoal, by burning which she had been accustomed to warm herself.

The color of the body is noteworthy and characteristic. The skin is bright red with a tinge of carmin, and the visible mucous membranes, especially the conjunctivæ and lips, are of a remarkably bright-red color. In order to show this better in the illustration they are somewhat everted. Like the skin, the internal organs are bright red in color, particularly those which normally are of a lighter hue. The blood in the vessels is cherry red and fluid, and this is especially noticeable in the meninges and brain.

The woman in falling from her bed received a cut on the bridge of the nose near its root. This is suffused with bright-red blood, which has also flowed over the forehead. Hemorrhage has occurred from the mouth and nose, and the blood has run in bright-red streaks over the adjacent parts of the face.

PLATE II.

ozone, or hydrogen. The [illegible] determining the [illegible]

[illegible]

ozone, or hydrogen. The cuprous solution is especially valuable in determining the carbon monoxid in illuminating gas.

Carbon monoxid may be burned to carbon dioxid, but this method is open to error through the presence of hydrocarbons.

Gruber[1] prefers to absorb carbon monoxid from air by blood, using Fodor's[2] method modified. From 10 to 20 liters of the suspected air are shaken with slightly diluted blood for fifteen minutes. The blood is then placed in a small flask and heated to boiling. Air, purified by being passed through a solution of palladous chlorid, is drawn through the heated blood and is conducted through solutions of lead acetate, dilute sulphuric acid, and finally palladous chlorid. A black precipitate in this indicates reduced palladium, and, therefore, the presence of carbon monoxid in the air tested. Fodor claims to be able to detect one part of carbon monoxid in 20,000 parts of air, in which he is corroborated by Gruber. Marsh-gas and other hydrocarbons do not interfere with the test as thus conducted. The test indicates quantities of carbon monoxid below the limit of toxicity. The quantity of palladium reduced may be determined by dissolving it in aqua regia and titrating with potassium iodid.

Vogel[3] uses diluted blood solution and examines the spectrum. Thus, by shaking diluted blood with from 100 to 200 c.c. of air and examining by the spectroscope, 2.5 per cent. may be detected. This represents a highly poisonous quantity. For lesser quantities 20 liters of air may be shaken with 10 c.c. of blood, which is then tested according to Fodor's method. The wash-water from the bottle in which the air was shaken may be used for the spectroscope, whereby 1 to 0.05 per cent. of carbon monoxid is detectable.

This latter test has the advantage of enabling the experimenter to submit the blood to further tests, yet of preserving the wash-water, with its characteristic action in the spectroscope, in a sealed tube as an exhibit.

Gas analysis apparatus of various types may be used for the analysis of larger quantities of carbon monoxid.[4]

Hempel[5] exposed mice to a contaminated atmosphere and examined their blood spectroscopically. By this means he was able to detect 0.5 per cent. of carbon monoxid, though his results are disputed by Gruber.

C. H. Wolf[6] has also given directions concerning the detection of this gas. For poisonous quantities, especially in the air of mines and other closed places, use is made of its physiologic action. A cage containing several varieties of warm-blooded animals is hung in the suspected atmosphere. If they die and the blood shows the presence of carbon monoxid, the test is definite. White mice are preferable to gray,

[1] *Op. cit.*, p. 145.

[2] J. von Fodor. *Deutsch. Vierteljahresschr. f. öffent. Gesundheitspflege*, vol. xii., pt. iii.

[3] *Ber. d. deutsch. chem. Ges.*, vol. x., p. 798; vol. xi., p. 235.

[4] L. M. Dennis, C. G. Edgar. "The Comparison of Rapid Methods for Determining Carbon Monoxid." *Jour. Amer. Chem. Soc.*, 1897, vol. xix., p. 859.

[5] *Zeitschr. f. anal. Chem.*, vol. xviii., p. 399. [6] *Pharm. Zeitschr.*, 1880, p. 268.

first, because they are accustomed to captivity, and, secondly, because the color of the blood is clearly visible in the ears, nose, and eyes, the last being especially brilliant.

Detection of Carbon Monoxid in Blood.—Since the discovery, by Hoppe-Seyler in 1862, of the spectrum of oxyhemoglobin, and in 1864 and 1865 of the spectrum of carbon monoxid hemoglobin, and its irreducible character, no toxicologic analysis of the blood has been considered complete without this test being applied. In forensic medicine it was first applied in 1867.

Blood taken from persons or animals poisoned by carbon monoxid shows a characteristic absorption spectrum closely resembling that of oxyhemoglobin. The absorption occurs between the D and E lines chiefly, and is apparent through the appearance of two absorption-bands of nearly equal intensity and width. The one near D coincides almost exactly with that of oxyhemoglobin of the same position; the

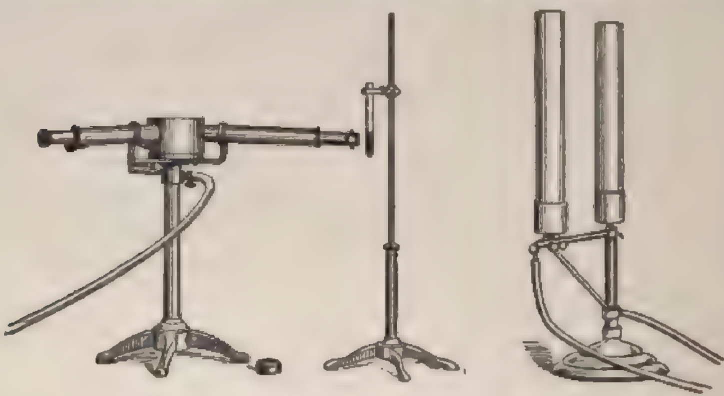


FIG. 76.—Spectroscopic examination of blood.

space between it and the band toward F is considerably wider than that between the two bands of the oxyhemoglobin spectrum (see No. 7, Plate 12). When more than 27 per cent. of the blood coloring-matter has been saturated by carbon monoxid, the addition to the blood of reducing agents, as a drop or two of a solution of crystallized ammonium sulphid, does not convert the spectrum into one of reduced hemoglobin. The character of this spectrum is that of a single band beginning on the D line and occupying a little more than half of the space to the E line (see No. 3, Plate 12). A solution of ferrous sulphate in ammonium tartrate may be substituted for the ammonium sulphid.[1,2,3]

The spectroscopic examination may be made by the use of an ordinary spectroscope, or, in a more refined manner, especially on very small quantities of blood, with the microspectroscope. The single prism spectroscope has an adjustable slit which is provided with a micrometer set-screw so that a particular width of opening may be accu-

[1] Hoppe-Seyler, *op. cit.*, p. 384. [2] *Med. chem. Untersuch.*, p. 202.
[3] W. Preyer, *Die Blutkrystalle*, Jena, 1871.

rately determined. Two burners placed on an adjustable stand supply white light—the one directly to the spectroscope, the other through a comparison prism attached to one-half of the slit. The blood contained in the test-tube is placed in the path of the rays from the first-mentioned lamp. The lamps are provided with tin casings which allow the light to pass through small windows cut in them. The position of any absorption-bands is determined by the illuminated scale of spectroscope. Recently methods have been used whereby photographs of the blood spectrum may be taken with suitable precautions to exclude optical delusions.[1] Dilution of the blood is advisable. Dreser[2] has made use of this method for the quantitative determination of carbon monoxid in blood. About one-third of the hemoglobin of the blood remains unconverted into carbon monoxid hemoglobin at the time of death under the toxic action of this gas on man and animals. Since some claim that carbon monoxid blood loses its characteristics when exposed to the air for a week, it is always advisable to seal up a specimen of the blood at the time of the autopsy. Such blood then retains its character for years.

Many prefer to extract the gas from the blood and measure and identify it. For this purpose 100 c.c. of the blood are introduced into the spacious receiver of a mercurial air-pump. The extraction of the gas is begun by the exhaustion of the blood by the action of a solution of glacial acetic acid saturated with sodium chlorid and preferably boiled, and the application of heat to about 50° C. By the use of proper absorbents, carbon dioxid and oxygen are removed, and the carbon monoxid can be measured by its absorption in cuprous chlorid solution. This solution may be caused to yield up its content of carbon monoxid by the addition to it of an excess of potassium hydroxid. The gas, if in sufficient quantity, may be ignited and seen to burn with a blue flame.[3]

The apparatus shown in the cut (Fig. 77), which is employed in the Toxicological Laboratory in Paris, may be advantageously used for extracting gases from blood. It consists of a container (*a*) of from 500 to 700 c.c. capacity, into which the blood is permitted to flow by means of the funnel (*b*) and stop-cock (*c*), the latter being large enough to allow clots to pass. Before the blood is let in the apparatus is exhausted by means of the mercury pump (*d*), aided by a water vacuum pump or by an air pump for quicker work. The gases disengaged from the blood are cooled in *e*, dried by the strong sulphuric acid on beads in *f*, deprived of carbon dioxid by stick potassium hydroxid in *g*, and finally carried to the holder (*h*). From this vessel they are delivered by aid of a capillary tube connected to the stop-cock *j* to a receptacle (not shown in the figure) placed in a pneumatic trough (*i*) holding mercury. The gases thus collected are then submitted to analysis.

[1] G. Bider, *Arch. d. Pharm.*, vol. ccxxx., pp. 280, 609–640.
[2] A. H. Dreser, *Arch. exp. Pharm.*, 1891, vol. xxv., p. 119.
[3] Brouardel et Ogier, *Documents sur les Travaux du Laboratoire de Toxicologie*, Paris, 1891, p. 26.
[4] *Ibid.*

The following additional tests are of importance because of their simplicity: Blood rich in carbon monoxid when boiled yields a brick-red mass; ordinary blood becomes brown-black (Hoppe-Seyler). Ten c.c. of 2 per cent. solution of a blood strongly charged with carbon monoxid, with 2 c.c. of yellow ammonium sulphid and 0.2 c.c. of 30 per cent. acetic acid, yields a bright-red, while normal blood yields a green, precipitate (Katagama). When blood is added to diluted hydrogen sulphid water drop by drop, it produces a bright-red precipitate when carbon monoxid is present, otherwise a bright green (Salkowski). Normal blood when shaken with one or two volumes of a solution of sodium hydroxid of 1.3 specific gravity becomes black, in thin layers dark greenish brown; carbon monoxid blood remains red and appears like red lead or cinnabar (Hoppe-Seyler). A mixture of cal-

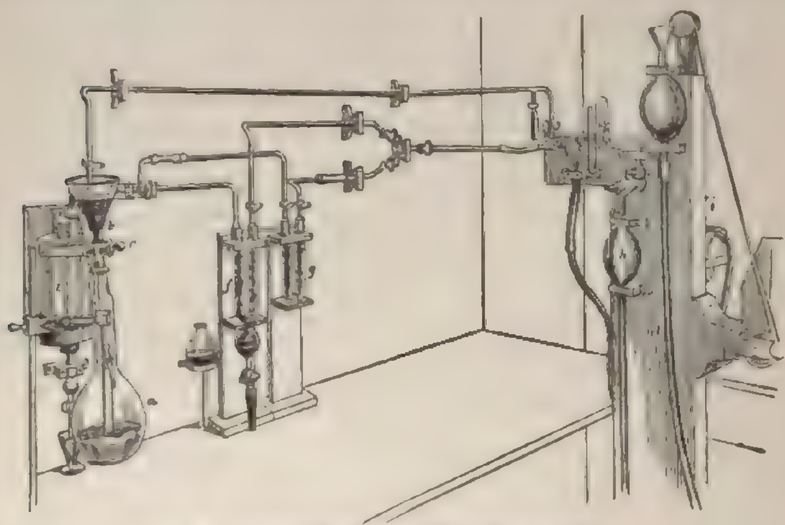


FIG. 77.—Apparatus for extracting gases from blood.

cium chlorid and sodium hydroxid produces a beautiful carmin-red color if carbon monoxid is present, while normal blood becomes brown (Eulenberg). A reducing agent, as ammonium sulphid, or alkaline stannous chlorid solution, or a ferrous salt changes the color of normal blood almost to black, while carbon monoxid blood retains its red color. Two c.c. of carbon monoxid blood with an equal quantity of water and three drops of a one-third saturated solution of copper sulphate when mixed produce a brick-red precipitate, while normal blood yields a greenish-brown one (Zallski). When four or five volumes of lead acetate are added to one of carbon monoxid blood and thoroughly shaken for about a minute, it retains its red color; normal blood, under like circumstances, becomes dark. The differences increase by keeping, being visible for a week or two even in open test-tubes (Rubner). When 10 c.c. of blood are mixed with 15 c.c. of 20 per cent. potassium ferro-

cyanid solution and 2 c.c. of moderately strong acetic acid (one volume of glacial acetic acid to two volumes of water), a solid coagulum gradually results; if normal, the coagulum is black-brown, but when the blood contains carbon monoxid, bright red (Wetzel). Blood diluted one part to four with water and shaken with three times its volume of a 1 per cent. tannin solution becomes in twenty-four hours of a gray tint when normal, but is carmin-red when carbon monoxid is present. The difference is still noticeable at the end of ten months (Wetzel). Some of the tests for detecting carbon monoxid in the atmosphere can be readily employed for detecting the gas when absorbed by the blood. Palladous chlorid can thus be made to indicate the presence and the quantity of carbon monoxid in blood.

POISONOUS GASEOUS MIXTURES.

Vapor from Burning Charcoal and Coal.—The three prominent factors in poisoning by products of combustion are diminution of oxygen, increase of carbon dioxid, and presence of carbon monoxid.

The vapors given off by the combustion of charcoal have been found to be highly poisonous, and though the carbon dioxid is in great proportion, the postmortem appearances and the symptoms indicate that the carbon monoxid is the more active agent.

From experiments by LeBlanc, a dog exposed to fumes of burning charcoal in a closed space succumbed when the air of the chamber contained carbon dioxid, 4.61 per cent.; carbon monoxid, 0.54 per cent.; carbureted hydrogen, 0.04 per cent.; oxygen, 19.19 per cent.; nitrogen, 75.62.[1] Biefel and Poleck have shown that charcoal fumes contain on an average a proportion of carbon dioxid to carbon monoxid of 20 to 1.[2]

The use of coal leads to many fatalities, since an improperly constructed damper or an open stove-lid permits the products of combustion to escape into the living-rooms. This form of poisoning is by far the most common of all from gaseous poisons. Little warning is given in these cases, since, as a rule, there is but a small quantity of sulphur dioxid which, by its suffocating odor, would indicate the gas to the occupants of the rooms.

The system of heating dwellings used abroad leads to many accidental deaths from the fumes escaping from the braziers. This form of portable stove is also too frequently a ready means at hand for suicide, especially since in foreign countries the sale of drugs is so carefully regulated. The mortality in France[3] due to this form of gas-poisoning far exceeds all other causes, and statistics from Germany[4] prove that this handy means has proved the most popular. Medical jurists recognize, however, in these matters the influence of suggestion.[5]

Symptoms.—The symptoms are not unlike those already described

[1] Taylor, *Medical Jurisprudence*, p. 446. [2] *Zeitschr. f. Biol.*, vol. xvi., p. 279.
[3] Hougonenq, *Toxicologie*, 1895. [4] Lesser, *op. cit.*, Berlin, 1888.
[5] Brouardel, *Les Asphyxies*, 1896.

for carbon monoxid. They have been described, indeed, by those who have succumbed to the action of the fumes—notably by Deal, who wrote of his sensations from the time of lighting the furnace at intervals of ten minutes until he became unconscious. A candle became extinguished, while a lamp continued to burn. The symptoms, from the beating of his temples to the madness that finally appeared, are all accurately described.[1]

The usual appearances of persons suffering from charcoal, coal, or coke fumes are: Lips purple; countenance livid; hands and nails purple; surface of body cold; breathing quick and short; pulse small, quick, and feeble; pupils fixed; total insensibility. There are cases, however, in which the face and skin are pale, eyes bright, and pupils dilated. There is often froth at the mouth or bleeding of nose or from lungs. Attitude generally composed; sometimes position shows an attempt to escape when overcome. There are sometimes contusions about head or body from precipitous falls. The bodies are, however, generally in a recumbent position, since most of the fatalities occur while the victims are asleep or have purposely assumed that position, having made preparation for self-destruction. When many are overcome, one or more may survive and show symptoms seemingly of drunkenness, or they are dazed, unable to answer questions, or, if able, answer incoherently; they are sometimes excited or delirious. These conditions frequently lead to a suspicion of murder, and there are many cases on record where the suspicions cast on the survivor have been dispelled only by a thorough investigation as to the cause of death. The fact that lamps, candles, or fires continue to burn is not evidence of the poisoning not being due to the products of combustion. At times the action of the vapors creeps on unawares.[2] In a recent case the escape of a large volume of furnace-gas from a defective flue into a church produced serious results. No one noticed the strong smell of gas. The congregation gradually became drowsy. During a lengthy closing prayer first one young woman and then another became unconscious. Others were made ill. The pastor became excited, and feeling strange sensations, called to the congregation to leave the church. All revived in the fresh air.[3]

From the time of Orfila[4] the French medicolegists have studied the various phases of this type of poisoning, and their literature abounds in cases covering almost every conceivable complication of circumstances and their medicolegal bearings.

An elaborate series of experiments on animals under the influence of charcoal fumes and the result of the pathologic findings are given by Biefel and Poleck.[5]

[1] Briand et Chaudé, *Manuel Complet de Médecine Légale*, Paris, 1858, p. 366.

[2] Brouardel, *op. cit.*, p. 29.

[3] Daily journals, November 30, 1897. Also report, W. P. Mason, *Jour. Amer. Chem. Soc.*, January, 1898.

[4] See Orfila, LeBlanc, Briand et Chaudé, Tardieu, Bugnion and De la Harpe, Devergie, Tourdes, Bernard, Brouardel, and others.

[5] Biefel and Poleck, "Ueber Kohlendunst," etc., *Zeitschr. f. Biol.*, Munich, 1880, vol. xvi., p. 279.

Treatment should be the same as for poisoning by carbon monoxid alone.

Postmortem Appearances.—These vary considerably according as the asphyxiation occurs through a preponderance of one or the other of the poisonous constituents of the fumes, coinciding either with those already given for carbon dioxid or with those for carbon monoxid. Even under nearly similar conditions of poisoning the postmortem appearances may vary. A comparison of two cases where death resulted from fumes of burning charcoal is given by Taylor.[1]

Anders reports a case of a physician overcome; his servant dead. The symptoms, treatment, and postmortem are given.[2]

Vapors from Lime, Brick, and Cement Kilns.—These are closely allied in composition and effects to the fumes from burning charcoal, coal, or coke. They are, however, given out in such quantities that, when cooled on their exit from the kilns by the surrounding air, they float almost bodily in certain directions, there being insufficient time for their diffusion. It thus happens that people sleeping near by are overcome, or that, by a sudden change in the draft, workmen are prostrated as if they had descended into a pool of the gas. Here again the postmortem appearances[3] incline either to those of carbon dioxid or monoxid, as already shown under the previous heading. Lime-kiln gases consist of carbon dioxid, 32 per cent.; carbon monoxid, 1.5 per cent.; oxygen, 1.5 per cent.; nitrogen, 65 per cent. Traces of sulphur dioxid and of hydrogen sulphid also occur if the fuel or charge contained sulphur.

It sometimes happens that, owing to the construction of the kilns, the gases are carried some distance underground before finding an exit. Their very variable composition may lead to the air being suddenly contaminated.

A practical and very instructive illustration of poisoning from these gases presented itself in 1887, at Malaunay, in the neighborhood of Rouen, where a certain Druaux kept a wine-shop in close proximity to a lime-kiln. Druaux and his wife's brother were found dead in the house, and Madame Druaux in a state of supposed intoxication. She was unable or unwilling to give any account of what had happened or any explanation of the fatalities, and was at once suspected of having administered poison. She was tried and found guilty. Her attorney was convinced of her innocence, and more by threats than by persuasion he succeeded in having her set at liberty after an incarceration of five years and nine months. Subsequently the matter was brought before the Court of Assize at Amiens, when the evidence then produced distinctly proved that carbon monoxid from the lime-kiln was not only the cause of the death of her husband and brother, but also of her

[1] Taylor, *op. cit.*, pp. 447–449.

[2] "Defective Furnace Arrangements," *Philadelphia Medical Times*, 1880, vol. v., p. 478.

[3] D. Herman, *Jour. Soc. Chem. Ind.*, 1896, vol. xv., p. 857; also same, p. 856.

supposed intoxication at the time. She was formally declared innocent, and awarded 40,000 francs as compensation for false imprisonment.

"After-damp" and Gases Left after Explosions of Gunpowder, Gun-cotton, etc., in Mines.—These also resemble the products of combustion already given. According to Haldane, after-damp consists of nitrogen and argon, 88.3 per cent.; carbon dioxid, 11.7 per cent.; while the gaseous mixture left along the track of explosions in coal-pits is carbon dioxid, 5 per cent.; carbon monoxid, 1.25 per cent.; oxygen, 12.5 per cent.; nitrogen and argon, 81.25 per cent. The argon is in the proportion of 1.18 in 100 volumes of mixed nitrogen and argon.[1] When sulphur gun-powder is used, sulphur and cyanogen compounds also result, while with nitro-powders, oxids of nitrogen are readily detected. Full details of appearances, symptoms, and pathologic findings are given in the voluminous special literature of mines, mining, and explosives.

"Water-gas," "Producer Gas," "Fuel Gas."—These technical names have been given to mixtures of combustible gases manufactured for general distribution or for special use in chemical and metallurgic works.

When steam is passed through red-hot coke, carbon monoxid and hydrogen should theoretically result; practically carried out, the process also yields carbon dioxid and marsh-gas. The resulting gas is odorless and very inflammable. When air is added, it forms a highly explosive mixture. Analyses of such gases are given by Herman.[2]

The great danger in the use of this gas by the public lies in its odorless character, its escape into the air remaining undetected; numerous casualties have resulted in which the histories disclose the fact that people were suddenly overcome while busy with the routine work of the house or trade.

The manufacture has been largely discontinued or modified to avoid these defects. In Europe the sale of such gas is interdicted. The better methods of gas-fitting practised in this country have lessened the fatalities.

A fierce discussion has brought out many facts regarding the use of these gases, all of which are to be found in technical literature and official reports.[3,4,5]

Symptoms, treatment, and **postmortem condition** are practically the same as in poisoning by carbon monoxid (see p. 652 *et seq.*). A few cases are selected from the numerous ones on record:

Two forge men, occupying a cabin where there was a cooking gas-stove supplied by water-gas, in the course of the day were found dead, as if asleep. The gas-cock was partially turned on. At the autopsy,

[1] Th. Schloesing, Jr., *Comptes Rendus*, vol. cxxiii., p. 302.

[2] *Jour. Soc. Chem. Ind.*, 1896, vol. xv., p. 857.

[3] S. W. Abbott, *The Relation of Illuminating Gas to Public Health.*

[4] W. T. Sedgwick and W. R. Nichols, "A Study of the Relative Poisonous Effects of Coal- and Water-gas." *Sixth Annual Report Mass. State Board of Health*, Boston, 1885.

[5] *Tenth Annual Report, Board of Gas and Electric Light Commissioners of Mass.*, Boston, 1895.

two days later, the bodies were examined by several medical men. Before the bodies were well opened the gas that escaped from them into the air of the room, of 39,000 cubic feet capacity and receiving 1000 cubic feet of fresh air a minute, was so contaminated as to affect several, —one seriously,—but all recovered.[1] The viscera presented the appearances of carbon monoxid poisoning, and the blood and viscera kept in stoppered bottles showed a rosy hue seventeen months later.

On January 6, 1887, owing to a break in the street mains in the city of Troy, N. Y., a quantity of fuel-gas passed beneath the frozen crust of earth and found its way into the adjoining houses. Three deaths and many more or less serious cases of illness resulted. The gas contained carbon dioxid, 5 per cent.; carbon monoxid, 37.5 per cent.; hydrogen, 48 per cent.; nitrogen, 7.1 per cent.; oxygen, 0.5 per cent.; marsh-gas, 0.9 per cent. The expressions of the deceased were placid. One victim, an old woman, was found seated in a chair holding her false teeth in her hand. The second, also a woman, lay upon the floor. The third, a man, sat upright on a lounge, his head reclining on his shoulder. The fire was burning in the stove, and the lamps were still burning on the table. Rigor mortis fully developed. Upon opening the cavity of the chest of one of the victims the physician bent forward and took one or two long whiffs for the purpose of detecting the presence of any odor. Almost immediately he was seized with giddiness and great oppression in the epigastrium, and was obliged to discontinue work for half an hour. The effects did not finally wear off until after twelve hours. Appearances of the organs those of carbon monoxid poisoning. A bottle of blood taken from the heart at the time of the autopsy was examined in February, 1888. The bottle had been closed with a tight cork. The blood had a strong odor of putrefaction, and was of a brilliant, vivid-red color, the same as noted at the autopsy. A few corpuscles were visible with the microscope. Characteristic absorption-bands were obtained with the spectroscope. The blood retained its character nearly two years from the date of the autopsy.[2]

In another case a father and two children were all taken sick right after dinner. When a physician came, he found that the father and one child had vomited freely. The child was drowsy, cold, and almost insensible. The father's head was cold, with perspiration on forehead. The other child was faint. The mother was sitting up, hands rigidly outstretched, able to talk, but at moments losing consciousness. All recovered by fresh-air treatment. They used a large fuel-gas stove, and it was found that it had been turned on and not lit—for how long is not stated. The fuel gas was made from anthracite coal and had a very slight odor.[3]

[1] Taylor, *Medical Jurisprudence*, p. 456.
[2] W. P. Mason, "Fatal Poisoning by Carbon Monoxid," *Jour. Amer. Chem. Soc.*, 1886, vol. x., p. 176.
[3] R. N. Flagg, *San. Eng.*, New York, 1882, vol. v., p. 39.

SATURATED HYDROCARBONS.

METHANE.

(Chemical formula. $CH_4 = 16$. Synonyms, "*Marsh-gas*"; "*Light Carbureted Hydrogen*"; "*Fire-damp.*")

This gas is a natural product forming the major portion of "natural gas" and the "fire-damp" of the mines. According to the report of the United States Geological Survey, the wells are located in the Middle States, and on January 1, 1896, there were still producing—in Pennsylvania, 1522; Indiana, 931; Ohio, 307.[1]

The uses of the gas are chiefly domestic fires and lighting by mantle systems, iron works, and glass works. "Fire-damp" is ever present in certain mines. In the Wilkesbarre anthracite region there is a constant outflow of gas that is piped to the surface and burns the year round. Accidents are frequent, and visitors are dissuaded from entering the mines. The gas mixes readily with air, owing to its lightness (specific gravity, 0.5596), and forms an explosive mixture as soon as it amounts to one-eighteenth of the volume of the air. Fortunately, the mixture does not ignite readily. Electrolytic gas—hydrogen and oxygen—ignites at 674° C.; marsh-gas and oxygen, at 656° C.[2,3,4] The force of explosion of the latter is 557 pounds per square inch, while marsh-gas with air gives 210 pounds per square inch. The miner's safety-lamp indicates by the "corpse light," or faint blue flame extending in the gauze cylinder, the presence of the gas before the proportion has reached the explosive stage.

As the gas has no odor, the miner is never warned of its presence by the sense of smell. It is only slightly soluble in water. It burns with a pale, illuminating, smokeless flame. In burning it yields watery vapor and carbon dioxid—"after-damp." It forms a large proportion of illuminating gas, and is present in "water-gas," "producer gas," etc.

Though generally considered an indifferent[5] gas, it has slight toxic properties. When mixed with air, it greatly reduces the proportion of oxygen; 45 per cent. marsh-gas with air leaves 11.5 per cent. oxygen and 43.5 per cent. nitrogen, while 70 per cent. reduces the oxygen to 6.3 per cent. and the nitrogen to 23.7 per cent. Haldane's experiments may be briefly put in tabular form and be thus compared with other gaseous mixtures. (See table on p. 667.)

While toxicologically it has little interest, from the fearful loss of life it annually causes through explosions in mines and as the result of escaping street gas it has a very important forensic bearing.

The extensive experiments relative to explosions in mines are closely related to the numerous explosions that have occurred in various cities since the introduction of steam heating and electric lighting, explosions

[1] *Amer. Gas-light Jour.*, 1897, vol. lxvii., p. 490.
[2] Victor Meyer, *Berichte d. deutsch. chem. Ges.*, 1893, No. 16.
[3] Freyer and V. Meyer, *Zeitschr. f. phys. Chem.*, vol. xi., pp. 1, 28.
[4] A. Mitscherlich, *Berichte d. deutsch. chem. Ges.*, vol. xxvi., p. 160; vol. xxvi., p. 428.
[5] L. Hermann, *Lehr. der exp. Toxikologie*, Berlin, 1874, p. 275.

that have resulted in the loss of many lives and of much property. Among these may be mentioned the serious one that took place some time since in Boston, where, during the construction of the underground road, a leak occurred followed by a disastrous explosion, causing extensive wreckage and great loss of life.

Carbon Monoxid (CO).		Carbon Dioxid (CO_2).		Fire-damp or Methane (CH_4).		Oxygen (O).	
Percentage present in air.	Effects on man.	Percentage present in air.	Effects on man.	Percentage present in air.	Effects on man.	Percentage present, remainder being N.	Effects on man.
0.05	After half an hour or more, giddiness on exertion.	3.5	Breathing deeper.	5.5	Nil.	17.3	Nil.
0.1	After half an hour or more, inability to walk.	6.0	Marked panting.	45.0	Breathing slightly deeper.	12.0	Breathing slightly deeper.
0.2	After half an hour or more, loss of consciousness and perhaps final death.	10.0	Severe distress.	70.0	Life endangered.	9.0	Breathing deeper and more frequent. Face bluish.
1.0	After a few minutes, loss of consciousness and final death.	15.0	Partial loss of consciousness.			5.0	Loss of consciousness and final death.
		25.0	Final death.			0.0	Death with convulsions.

The higher homologues of this series, C_nH_{2n+2}, are to be found in petroleum and are collected in the first distillates when cooled to a sufficiently low temperature. Other still higher members occur as gasolene, naphtha, etc. They have highly anesthetic properties, and have been used for such. Recently boys were detected in Philadelphia who climbed the lamp-posts, dipped rags in the naphtha of the reservoirs, and inhaled the vapors. They became mildly intoxicated. The effect was similar to liquor taken internally.[1]

UNSATURATED HYDROCARBONS.

ACETYLENE.

(Chemical formula, $C_2H_2 = 26$.)

This gas, present in very small proportion in illuminating and in oil gas, has recently acquired importance through the manufacture of calcium carbid by the aid of the electric furnace by the Willson process.[2] When wetted with water, calcium carbid changes to slaked lime and evolves acetylene. This ignites readily and burns with a brilliant but smoky flame. When burned from a properly constructed jet, it gives a very white light of greater intensity than any other known gas. A brilliant display was made of this at the Pan-American Exposition at Buffalo in 1901.

[1] *Amer. Gas-light Jour.*, 1897, vol. lxvii., p. 774.

[2] Rosemann, *The Mineral Industry;* A. H. Cowles, 1898, p. 75 *et seq.;* also Frank and Weil.

Acetylene is an endothermic compound. When a little fulminate of mercury is exploded in it, it detonates with violence, being decomposed into carbon and hydrogen (Berthelot). Mixed with a proper proportion of oxygen and ignited in an open mortar, it shatters the vessel (Victor Meyer). Mixed with air it also forms a violent explosive. Cyanogen compounds are formed as the result. It unites with metals forming acetylids, that of copper being the best known. These are also explosive. Acetylene may be liquefied at 0° C. under 26.05 atmospheres pressure. It is said to be the lightest liquid known, and has a high coefficient of expansion. This liquid is considered more explosive than the gas. In some countries liquid acetylene is classed as an explosive.

The gas has an odor of geranium, and is agreeable when pure (Moissan). Its escape into the air is usually detected because of the impurities. It is a product of all incomplete combustions of hydrocarbons, and is, therefore, found when lamps or gas-jets are allowed to burn with an insufficient air-supply. The characteristic odor of a Bunsen burner which has "retreated" or of an oil- or gas-stove-heated room is due mainly to acetylene.

It is also an intermediate product in the combustion of olefiant gas, and it escapes unburnt in the use of illuminating gas (Lewes). It is absorbed by water in sufficient quantity to impart a strong smell to the water and to yield precipitates with either ammoniacal cuprous chlorid or silver nitrate.

Very little was known of its physiologic action until recently, when experiments with large quantities showed that a considerable amount may be present in air without causing ill effects.[1]

The combination[2] with hemoglobin claimed to take place as with carbon monoxid and with nitric oxid, to form a bright-red compound which becomes gas free by the action of reducing agents, is denied by recent observers.

From experiments upon blood, acetylene has been found to behave as an indifferent gas.[3]

ETHYLENE.

(Chemical formula, C_2H_4 = 28. Synonyms, *Olefiant Gas; Heavy Carburetted Hydrogen.*)

This is a constant constituent of gas obtained by the destructive distillation of wood, coal, or oils. The "illuminants" of ordinary illuminating gas consist of a mixture of ethylene, propylene, and higher homologues, with which are also mixed members of other series, as benzene, naphtalene, etc. It has a pleasant odor, is slightly soluble in water, and unites directly with chlorin or bromin. The action of this gas and also of the other gases of this group is that of a weak narcotic. Gases of this category are now made by the destructive distillation of oil, and are

[1] N. Grehant, "Sur la toxicité de l'acétylène," *Comptes Rendus*, 1895, vol. cxxi., p. 564; L. Brociner, *ibid.*, 1895, vol. cxxi., p. 774.

[2] Liebreich and Bristow, *Ber. d. deutsch. chem. Ges.*, 1868, vol. i., p. 220.

[3] L. Hermann, *op. cit.*, p. 115.

then compressed in iron cylinders. These supply the gas for the Pintsch system of lighting.

Beyond their narcotic action, the other gases of this group have slight interest here.

ILLUMINATING GAS.

This is a mixture of various combustible gases, only certain of which, the "illuminants," burn with a bright, nearly white flame. The greater portion of the gas is hydrogen, with methane next in quantity. As the methods of manufacture differ and are continually changing, it is difficult to give an average analysis. Four or five varieties are delivered to consumers in New York. All observers agree, however, in ascribing its poisonous properties to the carbon monoxid it contains. Street gas deprived of carbon monoxid is not destructive to mice, though 11 per cent. be present in air (Gruber) and the air breathed for hours. Though stupefied, they recover promptly. Animals live in marsh-gas or hydrogen if supplied with sufficient oxygen (Freitag). Fortunately, the intense odor of street gas enables it to be detected long before the proportion of carbon monoxid in the air reaches the toxic limit. Tidy claims 1 part in 12,000 of air—certainly 1 in 8000; Soyka, 0.01 to 0.02 per cent., when the carbon monoxid would be only 0.004 per cent.; Gruber and others give the toxic limit of carbon monoxid as 0.02 per cent. The volume of gas lost to manufacturers by leakage is large—generally not far from 10 per cent.[1]

The retentive power of the soil on the odoriferous constituents of gas was accurately demonstrated in a long series of experiments, and many investigations bearing upon the subject of the escape of gas into dwellings through the soil were made by the workers in Pettenkofer's laboratory.[2,3]

An exhaustive experimental investigation concerning the permeation of the soil by gases showed that the ventilation *toward* dwellings was greater in winter than in summer, and the liability to poisoning in correspondence thereto.[4,5]

Cobelli gives the details of a case where the mother and two daughters were fatally poisoned.[6]

A father, mother, and daughter were overcome, in Berlin, by gas leaking from a pipe a distance of 30 to 35 meters.[7] A similar case is given by Wolffberg.[8]

[1] Th. Weyl, *Hyg. der chem. Gross-Indus*, Jena, 1896.

[2] Max von Pettenkofer, *Populare Vortrage*, 1877, pt. i., pp. 89-92.

[3] Biefel and Poleck, "Ueber Kohlendunst und Leuchtgasvergiftung," *Zeitschr. f. Biol.*, 1880, vol. xvi., pp. 312, 313, 314, 315.

[4] Welitschokowsky, "Experimentelle Untersuchungen ueber die Verbreitung des Leuchtgases und des Kohlenoxids im Erdboden," *Arch. f. Hyg.*, vol. i., p. 210.

[5] A. Suderkoff, "Ueber die Bewegung des Leuchtgases im Boden in der Richtung von geheitzten Wohnraumen," *ibid.*, 1886, vol. v., p. 166.

[6] Cobelli, "Vergiftung der Familie Caini durch Leuchtgas," *Zeitschr. f. Biol.*, 1876, vol. xii., pp. 420-433.

[7] Jacobs, *Berlin. klin. Wochenschr.*, 1874, p. 822

[8] S. Wolffberg, "Leuchtgasvergiftung nach Bruch des Strassenrohres," *Arch. f. Hyg.*, 1883, vol. i., p. 265.

Numerous experiments and many observations in cases of death from this kind of poisoning show that the toxic atmosphere may contain an insufficient quantity of gas to form an explosive mixture. It needs one volume of gas to twelve of air to explode. In greater proportion, as one to four, there is no explosion, but the gas takes fire.[1]

Street gas deprived of odor has poisonous properties nearly like those of water-gas or charcoal fumes. Indeed, a differential diagnosis between poisoning by illuminating gas and that by charcoal fumes is difficult to establish, even if the blood shows the characteristic carbon monoxid hemoglobin spectrum. This, however, has been attempted.[2] When symptoms of carbon monoxid are present in marked degree, illuminating gas is indicated. Sooty appearance of the mouth and nostrils tends to show charcoal fumes. Circumstantial evidence is more valuable than the pathologic appearances. Comparative experiments on animals are given by Biefel and Poleck.[3] It is difficult to diagnose a case of gas poisoning from one by alcohol or opium.[4] The character of the urine should be determined. It is often suppressed ; sometimes albuminous ; urea is sometimes absent.[5]

Symptoms.—Usually giddiness and headache, vomiting, loss of memory, unconsciousness, convulsions and loss of muscular power, and finally complete asphyxia.

As the number of cases, usually accidental or suicidal, is now very great, only a few can be referred to. One having a legal bearing of importance has been kindly communicated by Professor E. H. Bartley :

A workman in a gas-house was found dead at the bottom of a well in the gas-house where he worked. He was in the habit of going into this well as a part of his regular duties, going down a ladder. After his death it was claimed that on this day there was a leak in the pipes in the well, so that it was so full of gas that when he attempted to go down the ladder he was overcome by the gas, causing him to fall to the bottom, where he died of suffocation. On the other hand, the gas company claimed that there was no unusual leak in the pipes on that day, and that the man fell as the result of his carelessness or as a pure accident, and that his death was caused by concussion of the brain. The blood had a peculiar red color, and about two ounces of it were examined. Chemical and spectroscopic examination showed the blood to be thoroughly saturated with carbon monoxid. It retained its bright color for many days. From this fact it was concluded that the man had really died from gas-poisoning, and not from concussion of the brain or suffocation.

Treatment.—According to methods given under carbon monoxid.

Postmortem Appearances.—Coagulation of the blood. A deepened

[1] Biefel and Poleck, *op. cit.* ; Sonnenschein, *op. cit.*, p. 296 ; Dr. Schutt, *Vierteljahresschr. der ger. Med.*, third series, 1896, vol. vii., p. 162.

[2] J. Deichstetter, "Die gerichtlich-medicinische Differentialdiagnose zwischen Leuchtgas- und Kohlendunst-Vergiftung," *Friedreich's Blatter f. ger. Med.*, 1896, vol. lxxiv., p. 35.

[3] Biefel and Poleck, *op. cit.*

[4] John Norris, *Maryland Med. Jour.*, 1880–81, vol. vii., p. 341.

[5] McVey, *Medico-Legal Journal*, 1897, vol. xv., p. 270.

tinge of color. Pulmonary tissue brilliant red. Froth in air-passages and at the mouth. Engorgement of vertebral nervous system and extravasation of coagulated blood in the spinal column.

LITERATURE.

Kobert, *Intoxikationen*, Stuttgart, 1893, p. 535.
Koppel, *Inaug. Dis.*, Dorpat, 1891, pp. 120–136.
Maschka, *Handb. der ger. Med.*, Prag and Tübingen, 1882, p. 338.
Casper-Liman, *Handb. der ger. Med.*, Berlin, 1889, vol. ii., pp. 604–637, eighth edition.
Woodman and Tidy, *Forensic Medicine and Toxicology*, Philadelphia, 1887, pp. 486–488.

HYDROGEN SULPHID.

(Chemical formula, $H_2S = 34$. Synonym, *Sulphuretted Hydrogen.*)

This colorless, transparent gas, possessing the smell of rotten eggs, is found naturally in volcanic regions and impregnating many spring-waters. It is quite soluble in water, that of mean temperature dissolving 3.23 times its bulk of the gas. It is a product of the putrefaction of organic substances containing sulphur, and therefore always found where vegetable or animal matter is undergoing decay, as in cesspools, sewers, and privy vaults. It is also generated by the decomposition of organic matter by anaërobic bacteria in deep lakes and ponds, tainting the water or coming to the surface and tainting the atmosphere, where its presence is indicated by the discoloration caused to dwellings painted with white lead. Many of the small estuaries receiving the sewage of towns become defiled with this gas, being little more than open sewers. From such sources the gas is frequently discharged in considerable quantities, and is carried by the shifting winds in different directions. It is formed spontaneously whenever a soluble sulphate remains in contact with decaying organic matter with deficiency of air; directly by the union of sulphur and hydrogen; indirectly by the action of acids on the sulphids of the metals, generally ferrous sulphid. It is usual to prepare it in the laboratory from such sources as the latter. The gas is rarely pure. It is set free when sulphur is heated with damp wood, charcoal, tallow, or paraffin-wax. Its intense odor enables it to be recognized when present in minute quantities, 1 part in 10,000 being easily noted. It is slightly heavier than the air—specific gravity, 1.1912.

The solution in water gradually changes, depositing white sulphur. The gas is decomposed by heat into its elements. It is combustible, burns with a blue flame, producing water and sulphur dioxid, or, if the air is limited, some sulphur is deposited. It is decomposed in the presence of moisture by sulphur dioxid.

It explodes when mixed with half its volume of oxygen and ignited, with a deposition of sulphur; but two volumes of hydrogen sulphid and three of oxygen yield sulphur dioxid on explosion. It is one of the products of putrefactive processes in the body, especially in the intestine, and leads to auto-intoxication.

Symptoms.—Breathed in its pure state, this gas is immediately fatal.

It acts upon all animals through all tissues, especially the lungs. If somewhat diluted, it produces nausea, giddiness, cold skin, labored breathing, irregular action of the heart, pains in the stomach, and death by coma or in violent convulsions, with tetanus and even delirium. In greater dilution sleepiness will be produced, the continued respiration of the gas proving fatal, sensibility not being restored. In exceedingly dilute condition it sometimes occasions febrile symptoms somewhat resembling typhoid. Air containing 0.05 per cent. was the limit that men could breathe (Lehman). Sometimes symptoms appear after a considerable lapse of time after breathing the gas, and they may continue for some days. Sugar and urobilin have been found in the urine.

Chronic Poisoning.—Animals, as rabbits exposed to the gas or mice living in sewers, appear to become more or less habituated, and the same is true of men. Workmen in daily contact with the gas develop conjunctivitis, headache, and permanent gastric disturbances. The countenance becomes pale. The skin has a tendency to become furunculous. Chemists who frequently work with hydrogen sulphid are troubled with nervous headache in later years, and become exceedingly sensitive to this gas, which was at former times not unpleasant to them. Severe colic, "*plomb des fosses*," is brought on shortly after the inhalation of even very diluted gas—sometimes only a few whiffs. The depression of the nervous system is also distressing.

Besides its action on the nervous system, hydrogen sulphid has a specific action on the blood, changing the oxyhemoglobin to reduced hemoglobin and finally to sulphmethemoglobin, which is characterized by an absorption spectrum of two bands, one in the red midway between C and D, the other fainter, beginning at D and extending nearly half-way to E.[1] The blood does not show these in rapid poisoning. When inhalation has continued for some time, the bands have been detected, but must not be confounded with the same bands due to putrefactive processes.[2]

Lethal Dose.—The dose is between that of carbon monoxid and prussic acid—0.02 per cent. in the air may have toxic effect; 1 per cent. in the air would be destructive to human life (Letheby). Birds are especially sensitive, 1 : 2000 proving fatal, while only 1 : 200 kills dogs.

Postmortem Appearances.—Nothing characteristic where death has been instantaneous and the autopsy immediate. Later, such cases develop appearances as a brownish, viscid fluid in the nose and throat, and offensive odor from cavities and soft parts of the body. These odors give rise to unpleasant symptoms in those operating, and are more noticeable where the body has remained some time in the poisonous atmosphere. The muscles have a dark color and are irresponsive to electric stimulus; the lungs, liver, and other organs are distended with liquid blood; there is congestion of the right side of the heart, and the

[1] Hoppe-Seyler, *Centralbl. f. d. med. Wissensch.*, 1863, p. 433; also *Med. chem Unters.*, Berlin, 1866, vol. i., p. 151.

[2] L. Hermann, *Exp. Toxikol.*, p. 122. Full discussion of symptoms and of blood spectra, with citations of authorities.

body rapidly undergoes putrefaction. When poisoning has occurred from the highly diluted gas, the appearances are almost like those due to death from carbon dioxid. There is, however, a tendency to more rapid decomposition and a wide-spread green discoloration of the tissues of the abdomen. Indeed, the usual green tint of the abdomen in most cadavers is ascribed to the action of the hydrogen sulphid from intestinal gases on the blood coloring-matter diffused in the tissues.

Tests.—The odor is highly characteristic. Lead acetate paper, sensitive to 1 : 100,000 or in solution 1 : 250,000,[1] producing a brown or black precipitate or color. Cadmium and arsenic in hydrochloric acid solution produce yellow precipitates of sulphids. These are useful as quantitative tests. Solutions of hydrogen sulphid become blue when treated with $\frac{1}{50}$ of their volume of a solution of fuming hydrochloric acid, a few milligrams of para-amidomethanilin sulphate, and one or two drops of a dilute solution of ferric chlorid (Caro-Fischer test). The reaction is very sharp, 0.00009 to 0.0000182 grams of H_2S per liter being recognizable.[2] Boneko recommends this test in the analysis of urine.

A conclusive toxicologic analysis can be made only immediately after death and best at the place where the accident occurred. A bright silver or copper coin inserted in the muscles or mouth becomes tarnished. Lead-paper is blackened. Blood spectra are rarely obtainable.

SEWER AIR.

The poisonous effects of the air of sewers, cesspools, and privy vaults are due to hydrogen sulphid, ammonium sulphid, other undetermined gases, and to carbon dioxid and nitrogen. In many instances the asphyxiation is due solely to want of oxygen. The greatest danger is due to the presence of the sulphids. Paris sewer air gave on analysis in 100 parts—oxygen, 13.79; nitrogen, 81.21; carbon dioxid, 2.01; hydrogen sulphid, 2.99.[3]

According to French ordinances, 5 kilos of ferrous sulphate are to be added to each cubic meter of night-soil. The mass must be well stirred to expel gas. Many accidents result from not taking this precaution. In entering such an atmosphere a wet sponge should be worn or one of the types of respirator mentioned under carbon monoxid poisoning. In many works men must be so provided.

LITERATURE.

J. Pohl, *Arch. exp. Pharm.*, 1887, vol. xxii., p. 1.
K. B. Lehman, *Arch. f. Hygiene*, 1892, vol xiv., p. 135.
Koppel, *Litterar. Zusammenst. der von 1880–1890 in der Weltliter. beschrieb. Fälle von Vergiftungen von Menschen durch Blutgifte*, Inaug. Diss., Dorpat, 1891, p. 118.
J. Deichstetter, *Friedreich's Blätter der ger. Med. und Sanitäts-Polizei*, 1896, vol. lxxiv., p. 103
Th. Weyl, *Gewerbe Hygiene*, Jena, 1896.
Parke, *Hygiene*, Amer. edition, 1884, p. 146.
Woodman and Tidy, *Forensic Medicine and Toxicology*, Philadelphia, 1877, p. 494.

[1] Wormley, *Micro-Chemistry of Poisons*, second edition, Philadelphia, 1885, p. 372.
[2] Dragendorff, *Ermittelung von Giften*, fourth edition, Gottingen, 1895, p. 64.
[3] Taylor, *op. cit.*, p. 463.

SULPHUR DIOXID.

(Chemical formula, $SO_2 = 64$. Synonym, *Sulphurous Acid.*)

Found in nature in the vicinity of volcanoes. It is the product of burning sulphur or of metallic sulphids with air or oxygen. It frequently contaminates the air near metallurgic works, destroying vegetation. It is present in noticeable amount in the air of towns where coal containing sulphur is burnt. It is given off in the spontaneous combustion of coal. It is very noticeable in tunnels and underground railways, where the ventilation is poor.

In 1897 the whole population of Mount Vernon, N. Y., suffered from the suffocating fumes of the gas. The source was the improper purification of the illuminating gas. The air of homes and the streets was filled with sulphur dioxid.

It is produced in the refining of petroleum in large quantities. It is decomposed by sunlight into sulphur trioxid and free sulphur, thus producing part of the haze over large cities. It is readily oxidized to sulphur trioxid, which in its turn yields sulphuric acid when united to water. This acid causes destruction to property. Owing to the solubility of each of these oxids, the atmosphere is never found to have a considerable quantity of either. It is prepared either by the combustion of sulphur or by the deoxidation of sulphuric acid by charcoal or metals.

Workmen in many trades are constantly exposed to its action. Its intense odor fortunately prevents accidents.

It is an irrespirable, colorless gas, possessing the odor of burning brimstone. It is heavier than the air (specific gravity, 2.25). It is condensible to a clear liquid at –18° C., and is solid at –76° C. The liquid has a specific gravity of 1.45 at –20° C. and boils at –8° C. The liquid is sold in glass siphons or in metallic holders, and is much used in disinfection and artificial refrigeration.

The gas is very soluble, water absorbing 43.5 times its volume, and forming sulphurous acid, H_2SO_3.

Sulphur candles and liquid SO_2 are much used in domestic disinfection. The gas extinguishes flame even in the presence of much air. It is a powerful reducing agent. It is a preservative and a bleaching agent.

Symptoms.—The first are those of suffocation. The glottis closes against it. The air-passages are highly irritated through the formation of sulphuric acid on their surface. Coughing and sneezing are speedily induced. The remoter action is on the blood, reducing the same and decomposing it. Both men and animals may be habituated to its inhalation. Air containing from 0.03 to 0.04 grams per thousand did not affect workmen more severely than did from 0.01 to 0.02 those unaccustomed to breathing the gas.[1] Mice were affected more severely when 0.5 grain of sulphur was burnt in a receiver than when, after repeated exposures to such air, 10 grains were burnt in the same vessel.[2]

[1] B. K. Lehmann, *Arch. f. Hygiene*, vol. xviii., p. 180.
[2] Tidy, *op. cit.*, p. 495.

Letheby found the trainmen of the London Underground Railroad little affected by the constant breathing of the tainted atmosphere. Experiments on rabbits, mice, and guinea-pigs showed 0.04 vol. per cent., sufficient to bring on marked symptoms of poisoning, while 0.3 per cent. proved fatal.[1] Others claim that from 1 to 3 per cent. can be respired without ill effects (Hist).[2] The gas produced the same effect whether breathed through the nose or by tube introduced in the trachea. The cornea becomes opaque, and there are dyspnea, cyanosis, and convulsions. Taken internally, there are catarrh of the stomach and chronic sulphuric acid poisoning.

The effects on vegetation are greater than on animals. Suits for damages against works for the destruction of trees and other plants are frequent.[3]

Treatment.—Proper prophylactic measures: masks for workmen containing a wet sponge; ventilation; restriction of quantity used to preserve food or drink. Mild alkalis serve as antidotes.

Postmortem Appearances.—Those of asphyxia. The blood is very dark and has in part an acid reaction. The hemoglobin is changed first through loss of oxygen, then by decomposition, to hematin as with mineral acids. The respiratory tract is catarrhal or even croupous in aspect, due to the action of the acid. The lungs are partly edematous.

Tests.—Intense suffocating odor. Blue color with starch and iodic acid; sensitive to 1 : 3000. In foods and drinks it is tested for after being reduced to hydrogen sulphid by zinc and hydrochloric acid.[4]

NITROGEN MONOXID.

(Chemical formula, N_2O = 44. Synonyms, *Nitrous Oxid*; *Laughing-gas*.)

Laughing-gas is colorless, with a sweetish taste and smell. Water dissolves its own bulk. It supports combustion, but not life, as Davy supposed. It is eliminated from the blood unchanged. It has a specific gravity of 1.53. It becomes liquid at + 7° C. and 40 atmospheres pressure; solidifies at −102° C. at common pressure. It is sold in a liquid state in wrought-iron containers, and is used from these as an anesthetic. It is prepared for this purpose by heating ammonium nitrate, and must be carefully purified.

Symptoms.—When breathed in small quantity it produces a delicious tingling sensation and tends to induce laughter, hence its names, "paradise gas" and "laughing-gas." Persons differ greatly in what they do when under the influence of the gas at this stage. Its anesthetic effect on a young man, Cooley, who bruised his shins while dancing under its influence yet felt no pain, was noticed by Dr. Horace Wells, of Hartford, and led to its use in dentistry. Gardiner Q. Colton administered the gas to Wells at the latter's request, and Dr. Riggs extracted

[1] Ogata, *Arch. f. Hygiene*, 1884, vol. xi., p. 246.
[2] Th. Weyl, *Hygiene der chem. Gross-Industrie*, Jena, 1896.
[3] Schroeder and Reuss, *Die Beschädigung der Vegetation durch Rauch und die Huettenrauch-Schaden*, 1883; also Just and Blame, *Landw. Versuchsst.*, Jg 1889, pt. ii.
[4] L. Hermann, *Exp. Toxikologie*, Berlin, 1874, p. 137.

the first tooth without pain December 11, 1844. The gas fell into disuse, however, until 1863, when its use was revived by Colton. From February 4, 1864, the patients have inscribed their names on a scroll. The writer witnessed, January 17, 1898, gas administered to the 196,470th patient. No accidents have happened. By covering the nose and mouth with a respirator and keeping the patient's head slightly forward, the breathing is easy, unconsciousness speedy, the tint of the skin inclined to be blue, but not livid, insensibility complete, and recovery rapid. The use of the gas for prolonged surgical operations is growing, since there is rarely any subsequent vomiting. Access of air or 20 per cent. oxygen, or this mixture breathed under pressure, is employed, since nitrous oxid cannot replace oxygen for respiration.[1] The brain is first paralyzed, then the center of sensation of pain, then consciousness. The action then extends to the spinal cord, medulla oblongata, and finally to the heart. Asphyxia follows prolonged inhalation without oxygen. The blood-pressure is much augmented, and it is dangerous, therefore, to use it on patients with a weakened vascular system. Arterial blood shaken with the gas becomes dark, and venous blood, though shaken, does not become arterial. It does not combine with the blood, though this is claimed. Death during the administration has not been due to poisonous action of the gas, but to asphyxia or other cause.[2,3]

Treatment.—Fresh air, oxygen, and stimulants.

Postmortem Appearances.—No changes observable in man or animals even after many hours' narcosis except that the blood is of dark color.

Tests.—Solubility of the gas in alcohol. Explosion with hydrogen leaves a residue of nitrogen and water.

NITRIC OXID ($NO = 30$).

Also NITROUS ANHYDRID ($N_2O_3 = 76$); NITRIC PEROXID ($NO_2 = 46$).

Nitric oxid is a highly poisonous, irrespirable gas. It is colorless, nearly insoluble in water, but turns red in air giving N_2O_3, and NO_2, both of which dissolve. It is obtained by the deoxidation of nitric acid in voltaic batteries, or by the action of this acid on charcoal, metals, cotton, and vegetable fiber and by animal tissues. It is frequently evolved in great quantities in manufactures and laboratories, and must be especially guarded against by proper ventilation or by masks. Unfortunately, there is as yet no good absorbing respirator for these gases. Works, therefore, need constant supervision to protect employees.

Symptoms.—Habitually breathed in small quantities and great dilution it produces severe chronic diseases. In acute poisoning immediate dyspnea, tightness of chest, coughing, fainting, cyanosis, diarrhea, and collapse. Death within forty hours, though symptoms of slight poisoning are delayed, in which case the first symptoms are headache, desire for fresh air, thirst, and then suddenly symptoms of aggravated character—

[1] L. Hermann, *op. cit.*, p. 243.
[2] Kobert, *op. cit.*, p. 550; many citations of literature.
[3] Tidy, *op. cit.* (cases of death), p. 490.

distress of breathing, anxiety depicted on face, cold perspiration, protruding eyeballs, and spasmodic coughing, followed by vomiting.

Blood drawn on venesection tarry, thick, black, and rapidly coagulating. It is of diminished alkalinity, and on dilution becomes red and shows the oxyhemoglobin spectrum. Although Hermann [1] was able to obtain a definite compound of nitric oxid and hemoglobin, *ex corpus*, Belky [2] denies the possibility of its formation in poisoning by nitric oxid.

Treatment.—Removal of the person from the vitiated atmosphere; in commencing edema of the lungs administer atropin. When edema is absent, inhalations of vapor of water and a little ammonia. In cyanosis, alkaline salt injections.

Postmortem Appearances.—Congestion of the larynx and trachea and edema of the lungs. Brown-colored serum from any incision of the lungs. Veins of the pia mater full. The blood reacts acid.

Tests.—Starch and potassium iodid, after cautious acidulation with sulphuric acid, give blue color and detect traces. The color and odor of the gaseous mixture breathed are characteristic.

[1] L. Hermann, *op. cit.*, p. 112.
[2] Belky, *Virchow's Arch.*, 1886, vol. clx., p. 160; *Lehre der schädlichen Gase*, 1865.

FOOD-POISONING.

The term *food-poisoning* includes all forms of sickness caused by foods that contain organic poisons not added by human agency nor accidentally acquired through contact, but present in the tissues of the plant or animal during life, or developed during storage through decomposition changes wrought by various species of bacteria not connected with the usual diseases of man or animals.

Poisoning by Vegetable Foods.—Poisoning due to abnormal vegetable foods is not common, and, in this country, is of minor interest and importance. It is restricted practically to ergotized rye and solanized potatoes. The former has long been known as a cause of chronic poisoning in those parts of Europe where rye constitutes the principal food of the poorer classes, the outbreaks occurring most commonly after the use of grain harvested after wet, bad seasons; the latter have been known, within recent years, to cause numerous outbreaks of acute poisoning among soldiers of the German army. It has long been known that potatoes contain normally a very small amount (about 0.06 per cent.) of the poisonous principle, solanin, but it is only within recent years that it has been discovered that, under certain conditions, they may contain the poison in amounts sufficient to cause grave disturbance of the system. The increase is due to the action of at least two species of bacteria, *Bacterium solaniferum non-colorabile* and *Bacterium solaniferum colorabile*, and occurs in those tubers that, during growth, have lain partially exposed above ground, and in those that, during storage, have become well sprouted. The most extensive outbreak of potato-poisoning recorded[1] occurred in 1899 in a German regiment, fifty-six members of which, after eating sprouted potatoes, were seized with chills, fever, headache, vomiting, diarrhea, colic, and great prostration. Many showed more or less marked jaundice and several collapsed, but all recovered. Samples of the remaining potatoes yielded 0.38 per cent. of the poison, and this would indicate that a full portion must have contained nearly 5 grains.

The most important forms of food-poisoning are those due to unwholesome foods of animal origin, as milk, cheese, meat, and meat-products. In these the poisonous principles are products of bacterial action and may be divided into two classes—ptomains and toxalbumins.

Poisoning by Ptomains.—Ptomains are alkaloidal bodies produced by the action of certain micro-organisms, mostly unidentified, on proteid material; they are decomposition products and always contain nitrogen. To the lay mind the word conveys the idea of deadly poison,

[1] *Deutsch. med. Wochenschr.*, 1899, p. 783.

but all ptomains are not necessarily poisonous any more than all bacteria are necessarily pathogenic ; in fact, only a very small minority are poisonous, and these include mytilotoxin, obtained by Salkowski and Brieger from mussels ; tyrotoxicon, discovered by Vaughan in milk and cheese ; and cholin, neuridin, neurin, cadaverin, and putrescin, isolated by Brieger from decomposing meat and fish. Whether, in any case of decomposition, any ptomains will be formed, and if any, what kinds, depends upon the species of organisms present, the nature of the material, temperature, access of air, and other conditions. It appears to be not always essential that tissues containing ptomains shall be dead, for in a number of cases of poisoning following the ingestion of freshly gathered mussels and oysters the cause has definitely been demonstrated to have been these bases. As a rule, however, ptomain-poisoning is due to contaminated milk or cheese or to meats or fish that have begun to decompose. The stage of the process of decomposition is of no great importance, for as they are only transition products, ptomains may be present or may have disappeared at any stage, and meat that is not perceptibly tainted may be very toxic, while that which is decidedly rotten may be perfectly innocuous. In point of fact, most ptomain-poisoning is due to foods not markedly decomposed, since those that are repugnant to the senses are commonly rejected at once. It is possible that some cases of poisoning attributed to ptomains may be due really to albumoses formed in the first stages of decomposition of meats.

Ptomain-poisoning manifests itself, as a rule, within a short time after eating. The principal symptoms are those of more or less severe gastro-enteritis, but evidence of extensive involvement of the nervous system is by no means infrequent.

The first outbreak of poisoning traced definitely to a ptomain was that which led to the discovery, by Professor Victor C. Vaughan, of the intensely poisonous benzene derivative, tyrotoxicon, which he isolated from a number of cheeses, which, during 1883 and 1884, caused no fewer than 300 cases of sickness. The symptoms included abdominal pain, vomiting, diarrhea, feeble and irregular pulse, dryness and constriction of the throat, and cyanosis. Later the same poison was isolated by Wallace from a cheese that caused poisoning in 50 of the 60 persons who ate of it. In this outbreak the symptoms, which appeared within four hours, included vomiting, purging, griping, chills, severe epigastric pain, cramps in the feet and legs, and very marked prostration, and were most severe in some who ate the least. In 1886 Newton and Wallace[1] isolated the poison from milk that caused extensive outbreaks in several hotels at Long Branch. In all, 73 guests were affected. They were seized soon after supper with nausea, vomiting, cramps, dryness of the throat, burning sensation in the esophagus, and collapse. Some had active diarrhea with no vomiting, and some had no diarrhea. As a rule, nausea and vomiting were persistent and were accompanied by a tendency to exhaustion and collapse.

The most remarkable case of milk-poisoning on record was investi-

[1] *Medical News*, September 25, 1886.

gated by Professor Vaughan. It involved four persons, three of whom died. The symptoms were essentially the same in kind, but differed somewhat in severity. They included severe vomiting, flushed face, labored breathing, burning constriction in the throat, dysphagia, dilated pupils, subnormal temperature, and stupor. In the three fatal cases death occurred in four, five, and six days, respectively, from the onset. In the person who recovered improvement began about five days after seizure, but complete restoration to health required a month. The milk that was at fault had been kept in a buttery that was in a most unsanitary condition. Fresh milk stored therein overnight and then examined gave both chemical and physiologic evidence of tyrotoxicon, as did also portions of milk inoculated respectively with dirt from the floor, vomitus, and aqueous extract of the intestines of the victims.

As an example of poisoning by another ptomain may be cited an outbreak caused by mussels. This occurred in 1885 at Wilhelmshafen, Germany, and involved 19 persons, who were seized shortly after a meal of freshly gathered mussels with very severe symptoms, which included nausea, vomiting, constriction of the throat, dizziness, trembling, diminished coördination of movement, dilated and unresponsive pupils, difficult speech, and coldness and numbness of the extremities. Very soon the legs became so weak that the patients could not stand. Abdominal pain and purging were absent. Four deaths occurred: one within two hours, one in three and one-half hours, and two in five hours, after eating. Specimens of the mussels were examined by Salkowski and Brieger, who isolated therefrom a ptomain to which they gave the name *mytilotoxin*.

In most cases of ptomain poisoning the tongue is coated, there is great thirst with profuse sweating and salivation, and, if the central nervous system is involved, the evidence is restricted to disturbance of sight—dilated pupils, diminished reaction to light, and rarely, amblyopia and diplopia. Ptosis, which is very commonly observed in the forms of food-poisoning yet to be considered, is never observed in ptomain poisoning.

The postmortem appearances are usually those of more or less severe gastro-enteritis. The mucous membrane of the intestinal tract may show almost nothing abnormal or may be extensively changed. Hemorrhages may or may not be observed in the intestines and other organs. In many cases the brain is anemic, and often there is edema of the lungs.

Poisoning by Proteid-Toxins.—Poisoning by proteid toxins is of two kinds: (1) That due to toxins produced by certain species of bacteria, chiefly anaërobic, in meats and fish originally wholesome; (2) that due to toxins present before slaughter in the tissues of animals suffering with septic disease.

The first kind includes that form which, for many years, has been known as *allantiasis* or *botulism*, from the fact that hundreds of cases were traced to sausages (*botulus*, a sausage). In view of our present knowledge of the subject the term is a misnomer, for sausages are by no means the sole cause. Indeed, the outbreak that led to the discovery

of the specific organism (*Bacillus botulinus*) and its toxin was due not to sausages, but to pickled ham; and the same train of symptoms has been caused repeatedly by fish.

In botulism the onset occurs, as a rule, between twelve and twenty-four hours after eating. The shortest recorded time of onset is thirty minutes; the longest, nine days. As a rule, the symptoms begin with general discomfort, eructations, nausea, vomiting, diarrhea, and abdominal pain, amounting sometimes to severe colic. After a time the diarrhea ceases and is succeeded by obstinate constipation. Within two or three days symptoms of more or less profound poisoning of the central nervous system make their appearance, involving especially the sense of sight. They include dilated pupils, paralysis of accommodation, amphodiplopia, amblyopia, ptosis, and glossopharyngeal paralysis. The mouth is dry, there is a sense of burning constriction in the throat, with great thirst, which cannot be relieved on account of inability to swallow. The voice is hoarse for a time, and then is wholly lost. All the secretions are diminished, and some are suppressed. The skin is dry and cold; the temperature usually subnormal, but sometimes high. The pulse is small and rapid, but may be slow. Exhaustion is extreme, and the slightest movements are made with great difficulty. As a rule, consciousness is preserved throughout, but in some fatal cases light delirium and coma have come on a few hours before death. In case of recovery the nervous symptoms gradually subside and convalescence is very slow. During convalescence desquamation of the skin and falling of the hair are not uncommon, and relapses may occur at long intervals.

Among the less common symptoms are acute nephritis, paralysis of the bladder, drowsiness, insomnia, dizziness, numbness, cramps, convulsions, and paralyses.

Not all the symptoms mentioned are necessarily observed in every outbreak; but in any single outbreak the common cause excites in all the victims a train of symptoms in rather close agreement, the differences being due to idiosyncratic causes or to differences in the amount of the poison ingested. Doubtless, too, the general condition of the system, and more especially the condition of the mucosæ of the digestive tract, are not without influence.

As an example of the tendency to relapses, the following case, reported by R. David,[1] may be cited. Each of five adult members of a family ate a whole raw red herring in the beginning stage of decomposition, but all did not suffer to an equal extent. In two days the son was seized with loss of appetite, disagreeable eructations, diarrhea, vomiting, dryness of the throat, and general weakness. Improvement occurred after twenty-four hours, but was followed by a change for the worse. The diarrhea was succeeded by obstinate constipation, which yielded finally to cathartics. Five days later the sight began to be affected. First, there was dimness of sight, which, after a week, was followed by double vision. Coincidentally, he experienced great difficulty in swallowing. Then the effects began to subside, and in about

[1] *Deutsch. med. Wochenschr.*, 1899, No. 8.

ten weeks he recovered. The mother, aged sixty-seven, and the father, aged sixty-five, were seized respectively on the fifth and ninth day after eating with practically the same symptoms that the son had shown. Both recovered in six weeks. The daughters were less fortunate. One was seized on the third day with bad taste in the mouth, constipation, and dryness of the throat, and six days later with dimness of sight, double vision, paralysis of accommodation, and dysphagia. Five weeks later, owing to inability to swallow, food was introduced by means of the stomach-tube. There was ptosis of the right eyelid, then of both; the voice was nasal; the pulse very small, but not very rapid. A week later the bladder became paralyzed, and in a few days there were additional evidences of bladder and abdominal disturbance. Then improvement set in, and in the course of six or seven weeks she felt perfectly well; but a month later she suffered a relapse with reappearance of the original symptoms, which persisted in some degree for another month. About seven months from the time the trouble began she recovered completely. The other daughter was seized on the seventh day with symptoms that were more severe and varied than those of her sister. Improvement began in about six weeks, but she then began to show alternate gains and losses of ground, and in a few days a scarlatiniform eruption appeared over the whole body, accompanied by albuminuria. Severe pains occurred in both hypochondria, with tenderness in the region of the kidneys and epistaxis, followed by disappearance of the rash, slight desquamation, and improvement of vision. Two weeks later the tenderness in the region of the kidneys and the albuminuria disappeared, and swallowing became normal; but at the same time cardiac complications occurred, which lasted five months, ending in hypertrophy of the heart. In the meantime she suffered a relapse like that of her sister, after a general improvement. Eight months from the onset recovery was almost complete.

In this case, owing to the fact that none of the offending food was available for examination, no bacteriologic examination was possible, but, from the similarity of the general symptoms to those of an outbreak investigated by Van Ermengem,[1] it seems probable that the cause of the sickness was the same organism that he isolated and named *Bacillus botulinus*. This he obtained from a portion of a raw pickled ham that poisoned 20 persons, 3 of whom died. Part of the ham and other meat from the same animal had been eaten from time to time before without causing any ill effects. That which caused the outbreak was the only part which, during the months that had elapsed since slaughter, had been immersed completely in the pickling brine. It presented no abnormal appearance, considering the length of time it had been in pickle, and gave off no odor suggestive of putrefaction; but it had a rancid smell and an unpleasant taste. The uneaten part yielded great numbers of an anaërobic bacillus, which also was found in the spleen of one of the victims. The meat itself and aqueous extracts thereof produced in animals the same train of symptoms as had been caused in the

[1] *Zeitschr. f. Hyg. u. Infektionskrankh.*, vol. xxvi., p. 1.

consumers. As a rule, the experiments ended in death. The specific toxin was isolated, and its study gave most interesting results.

Autopsy of two of the victims of this outbreak showed hyperemia of the kidneys, liver, and meninges, and extraordinary friability of the stomach-walls. In one case the liver showed marked fatty degeneration, and the brain, punctiform hemorrhages. The liver and kidneys yielded negative results on bacteriologic examination, but the spleen contained great numbers of the bacillus above mentioned.

Not all cases of toxin-poisoning due to bacterial contamination of originally wholesome meats fall under the head of botulism, for other bacteria than the anaërobic *Bacillus botulinus* can produce toxins that may cause equally severe symptoms of a less varied character, without such extensive involvement of the central nervous system. The different species of Proteus, for example, elaborate powerful toxins that have been known to cause fatal poisoning, of which the following cases may serve as examples. In an outbreak reported by W. Silberschmidt[1] 50 persons were made sick by sausages made of pork and cow-beef that had appeared to be of good quality when the sausages were prepared. The method followed in making them, however, was such as well might lead to decomposition and invite contamination. The particular form of sausage made, which is known as the "Landjäger," is highly esteemed by the poorer classes in Switzerland and elsewhere, and is made in the home. The meats, commonly beef or horsemeat with pork-fat, are chopped coarsely, spiced, stuffed into casings, and then pressed flat for a day. Then they are smoked for two days and dried in the air for several days, at the end of which time they are ready for consumption, uncooked. Not uncommonly they hang in the chimney several days, being smoked and heated by day and freezing by night. While the alternate heating and freezing cannot but be favorable to decomposition, the smoking serves to mask any disagreeable odor or taste arising therefrom.

As soon as this particular lot of sausages was ready for sale they were disposed of, and those who ate them were soon made sick. The chief symptoms were as follows: Very severe, partly crampy, abdominal pains; very profuse diarrhea, the stools numbering from eight to twelve a day, and in color, gray, greenish, and yellow; vomiting of brownish watery matter; sunken eyes; great lassitude; tenderness over the abdomen; cramps in the calves; great thirst and high fever. In general, the symptoms appeared on the day after eating. Some of the victims recovered in a day, some as late as a month, but the majority were sick a fortnight and were fit for work in three weeks. In one case there was a relapse: the victim was sick eighteen days and then returned to his work, but was seized again with the same train of symptoms ten days later. One man who ate two whole sausages suffered no inconvenience whatever beyond slight pain on the following day.

One young man of eighteen years died two and a half days after eating. When seen, he was passing grayish, watery stools, and his abdo-

[1] *Zeitschr. f. Hyg. u. Infektionskrankh.*, vol. xxx., p. 328.

men was very sensitive; pulse very small, irregular, and rapid. Some hours later he was delirious, and during the night he collapsed and died. Autopsy showed a spleen of normal size, swollen mesenteric glands, and hyperemia of the stomach and intestines. The follicles were much swollen, and in the ileum were several areas of erosion. The other organs were normal. Chemical analysis of the remaining sausages showed nothing noteworthy. Bacteriologic examination showed the presence of a number of species of bacteria, as was but natural, the most prominent being *Proteus vulgaris*.

Another outbreak that involved seventeen persons, one of whom died, was traced by E. Levy[1] to Hauser's proteus. The symptoms included severe, bloody diarrhea, meteorism, cramps in the calves, and slight fever. The discharges yielded a virulent culture of the organism above mentioned. All the victims had eaten in a restaurant where the meats were kept in a refrigerator, the bottom of which was covered with a slimy brown coating that had an unpleasantly sweetish smell, and yielded a virulent proteus culture. The several cultures gave with animals the typical picture of septic poisoning, and sterilized cultures produced the same results.

There are certain points of resemblance between the symptoms of botulism and those of poisoning by belladonna, stramonium, and hyoscyamus, but there are also certain marked differences that simplify the diagnosis. Thus, in poisoning by belladonna and allied plants, the onset is rapid, the face is flushed, consciousness is lost, there may be delirium and hallucinations, and the pulse is very rapid. In gelsemium poisoning, too, there are certain resemblances, but it runs a short course to death or recovery, the disturbance of accommodation soon passes, and the secretions are not affected. In poisoning by conium and hellebore there is marked salivation instead of dryness of the mouth, and in the former, paralysis of the lower extremities occurs, and is followed by paralysis of the upper extremities; and in the latter, there are convulsions, delirium, stupor, and collapse.

The postmortem appearances in botulism include nothing characteristic. Cadaveric rigidity appears often very early and may be very persistent or may be slight and transient. Commonly there is marked hyperemia of the various organs and especially of the meninges, bronchi, lungs, alimentary tract, and liver. The mucous membrane of the stomach is frequently injected and spongy. The spleen is sometimes enlarged and extraordinarily soft and friable. The liver sometimes shows fatty degeneration, and the kidneys, hyperemia and small infarctions.

The second kind of poisoning by proteid toxins is due to bacteria and their toxins present in the tissues of animals during life. It was pointed out as early as 1876 by Bollinger that a very large proportion of cases of food-poisoning was due to eating the flesh of animals suffering with pyemia and septicemia, and his statement holds good to-day. In the countries of Europe—and it is there that meat-

[1] *Arch. f. exper. Path. u. Pharm.*, vol. xxxiv., p. 342.

poisoning is most frequently observed—the peasantry and poorer classes in general have not that abundance of animal food that obtains on this side of the Atlantic, and when a domestic animal is sick, apparently beyond recovery, there is an incentive to take advantage of the opportunity to put it to a useful purpose rather than suffer a total loss. Natural death is, therefore, forestalled by slaughter, and the meat is sold for what it will bring. Many outbreaks have been traced to this utilization of sick cows with septic disease following parturition and of calves with specific enteritis and various pyemic and septic diseases. Van Ermengem has suggested that a number of septic diseases of these animals are grouped commonly under the general heading of diarrhea, and he has obtained positive results on feeding the flesh of one so suffering to mice and other animals. He has succeeded also in isolating a bacillus resembling Gärtner's *Bacillus enteritidis* from the bone-marrow, and this, on inoculation, was found to cause a fatal enteritis. In a large number of outbreaks of poisoning by diseased meat bacilli of the coli group, which produce toxins endowed with remarkable resistance to the influence of high temperatures, have been isolated.

The symptoms of this class of cases range from loss of appetite, vomiting, diarrhea, abdominal pain, headache, prostration, fever, and chills, in mild cases, to most profound disturbances, often resembling the general picture of Asiatic cholera. The discharges are sometimes watery and greenish, sometimes like those of typhoid fever and cholera. Very frequently the first stools are extremely offensive. Sometimes the case resembles acute arsenic poisoning, but the purging commonly precedes vomiting, while in arsenic poisoning this order never obtains. Fever is another diagnostic point, but in septic meat-poisoning the temperature is not necessarily elevated. Again, in arsenic poisoning, the abdominal pain is violent and there is burning pain in the epigastrium. Often the disorder is mistaken for typhoid fever, which it may closely resemble.

Sometimes the onset is very much delayed. In one large outbreak the majority of victims were seized within a week, and the remainder during the second and third weeks. Illustrative of this class of poisoning, the following cases are given:

A cow suffering with violent diarrhea and high temperature was slaughtered, declared unfit for food, and ordered destroyed. The carcass, instead of being destroyed, was stolen and made into mince meat, which was eaten raw by 80 persons, all of whom, even those who ate but little, were seized in from three to sixteen hours with nausea, vomiting, and diarrhea. Some had high fever. The meat was examined by Kaensche,[1] who isolated therefrom a bacillus (*Bacillus Breslaviensis*) which proved to be pathogenic for animals.

Van Ermengem [2] investigated an outbreak involving a large number of persons who ate the flesh of two sick calves. The symptoms were chiefly those of gastro-enteritis—severe vomiting and diarrhea, fever,

[1] *Zeitschr. f. Hyg. u. Infektionskrankh.*, vol. xxii., p. 53.
[2] *Bull. de l'Acad. Royale de méd. de Belgique*, 1892, p. 1025.

and great prostration. From the spleen, liver, and intestines of those who died and from the marrow of one of the calves he isolated a bacillus (*Bacillus Moorseelenensis*) much like, but not identical with, Gärtner's *Bacillus enteritidis*. Cultures thereof sterilized by heat caused in animals the same train of symptoms as was produced by living cultures.

An outbreak at Cotta, Saxony, investigated by Johne,[1] in which 137 persons were seized, was traced to meat from a cow slaughtered, in consequence of an inflammatory condition of the udder, six days after suddenly ceasing to give milk. The meat appeared to be normal, and was sold on the day after slaughter. During the night of the day of sale the first cases occurred, and soon the number reached 137. In the majority of cases the disorder began with nausea, vomiting, diarrhea, headache, abdominal pain, intense thirst, restlessness, and great lassitude. In one case there was a premonitory chill. In another, the symptoms began with difficulty in swallowing, double vision, and anxiety. In many cases the eyes were glassy and the pupils widely dilated. The tongue was commonly dry and coated. Many of the victims were children, all of whom were extraordinarily weak, and some had fever as high as 104.7° F. The meat was eaten raw by the majority, and either cooked or as broth by the rest. Johne isolated a bacillus much like Gärtner's from the meat and from the heart, spleen, and intestinal contents of one of those who died. In the meat it was not found in the small blood-vessels, but alone or in chains and clumps between the fibrillæ.

The well-known Mansfeld case, reported by Wesenberg,[2] and quoted extensively as a striking instance of the danger of eating the flesh of sick animals, is one which admits of considerable doubt as to whether the trouble caused was due to the condition of the meat at the time of slaughter or to contamination thereafter, inasmuch as those who ate first of the meat did not suffer, and that which caused the outbreak was stored in a cellar where it had every opportunity of becoming infected by Hauser's proteus. The meat was that of a cow slaughtered in consequence of traumatic pericarditis. Those who ate of the cooked meat escaped the ill effects that were suffered by those who ate the raw meat or the partly cooked liver. The symptoms caused were as follows: Vomiting, diarrhea, violent headache, abdominal pain, general muscular weakness, dizziness, and lassitude. The discharges were greenish or brownish, and always extremely offensive. With few exceptions the symptoms abated within from three to five days, and all the victims recovered, with, perhaps, one exception—a child who was not known with certainty to have participated in the feast. Inoculation experiments on animals with bouillon cultures of the unconsumed meat produced fatal results, and autopsy showed enlargement of the spleen, marked injection of the small intestine, and redness of the medullary substance of the kidneys.

[1] *XXI Jahresber. u. das Medicinalwesen im Konigreich Sachsen*, p. 104.
[2] *Zeitschr. f. Hyg. u. Infektionskrankh.*, vol. xxviii., p. 484.

Poels[1] has reported an outbreak due to meat from an apparently normal cow, consumed by 51 families, members of 24 of which suffered, while those of the other 27 escaped. The unconsumed meat appeared to be normal in every way even after eight days, but it yielded bacilli, apparently Gärtner's *Bacillus enteritidis*, which killed susceptible animals with symptoms of paralysis and intestinal catarrh. The organs of the experimental animals were poisonous, even when boiled, and the toxin was found to act with undiminished virulence on long boiling. Sterilized and living cultures caused the same symptoms in calves.

The first case in which the organism known as Gärtner's bacillus was proved to be the cause is that known as the Frankenhausen[2] outbreak, in which 58 persons were made sick after eating the flesh of a cow slaughtered in consequence of severe diarrhea. The symptoms were, in general, nausea, vomiting, diarrhea, drowsiness, dizziness, pains in the extremities, and fever that in some instances reached 104° F. All who ate of the raw meat were seized without exception, and the severity of each attack was directly in proportion to the amount eaten. One man who ate 800 grams was seized within two hours and died on the following day. Those who ate of the cooked meat fared differently, for they did not suffer directly in proportion to the amount eaten, and, indeed, some escaped entirely. Some who ate much suffered little, and some who ate little suffered much. Some who ate nothing but broth suffered severely, while the number of those who ate the cooked meat without injury was 36.

From some of the uneaten meat and from the spleen of the one fatal case Gärtner isolated the bacillus, *Bacillus enteritidis*, which has been proved to be the exciting cause of many later outbreaks. In almost every experiment it caused sickness, and many of the animals used died. The cooked meat and the broth also produced like effects. The organs of the animals showed myriads of the bacilli in the smallest blood-vessels, and the bacilli were shown to produce a toxin that resists the boiling temperature. In this outbreak nearly every victim underwent extensive desquamation of the skin, even of that of the horny surfaces of the hands and feet. In the well-known case of Gaffky and Paak[3] there was probably a double poisoning: first a ptomain and then a toxin poisoning. At least 30, and probably more, persons were made sick by horse-meat and horse-sausage. The symptoms came on shortly after eating,—in one case within a half-hour,—and comprised nausea, headache, abdominal pain, borborygmus, diarrhea, dizziness, trembling, and great thirst. The temperature rose to 104° F. Recovery occurred within a short time. Examination of the meats revealed a bacillus that bore certain resemblance to Gärtner's.

Gärtner has pointed out that not all parts of a diseased carcass are equally poisonous, and that in some organs the specific bacteria may be very numerous and in others they may be relatively scarce. The

[1] *Weekbl. v. h. Ned. Tijdsch. v. Geneeskunde*, 1893, vol. ii., No. 3.
[2] *Correspondenzbl. d. allg. aerztl. Vereins von Thüringen*, 1888, No. 9.
[3] *Arbeiten aus dem kaiserlichen Gesundheitsamte*, vol. vi., p. 159.

former must, therefore, be the more poisonous. The bacteria are, moreover, capable of multiplication after slaughter if favoring conditions, as of temperature, are present; and thus a few present in the tissues of the animal during life may increase to many during storage after death.

In many cases of meat-poisoning in which the character of the offending material was such that no evidence could be adduced to show that the animals from which it came were diseased, bacilli have been isolated that were identical with, or very similar to, that which so often has been found in cases of outbreaks due to septic meat,—*i. e.*, Gärtner's *Bacillus enteritidis*,—and illustrative of this, the following cases are cited: Karlinski[1] has reported the case of a man who, on a wager, ate 400 grams of meat that had been dried in the open air and exposed for months to wind, dust, and rain. In two hours he was seized with nausea, vomiting, diarrhea, headache, and fever. During convalescence desquamation of the skin occurred on the neck and extremities. From the vomitus and stools Karlinski isolated a bacillus that bore a close resemblance to Gärtner's.

Dr. W. N. Parker[2] investigated and reported an outbreak of poisoning that occurred, in 1899, at Sheffield and involved 24 persons, ranging in age from two to eighty-nine years, of whom one died, after about five hours, of convulsions. The cause was canned corned-beef, which appeared to be somewhat soft, but was devoid of odor and abnormal taste. All those who ate of it were seized with symptoms that, in general, were the same, although in individual cases there were some slight peculiarities. The following may be taken as a fair example: A woman of thirty-five ate two ounces of the meat, and in two hours was seized with sudden faintness, dizziness, and drowsiness, followed by nausea and great muscular weakness, especially of the legs. This condition was followed shortly by persistent vomiting and retching, intense frontal headache, colic, and purging. An hour later she collapsed. The face was pale and bathed in sweat; the skin was cold and clammy; pulse, small and rapid; respiration shallow; temperature subnormal; pupils dilated. After her stomach had been washed out, her general condition improved, but within an hour she collapsed again, and the original symptoms reappeared. After a second washing out she again improved, and on the following day she had only a slight frontal headache.

The only fatal case was of a boy of seven who ate about two ounces. His symptoms were especially severe, collapse was profound, and he required constant stimulation. Ten hours after the onset clonic contractions of the flexor muscles of the neck, arms, and legs occurred. These were violent, rapid, and almost rhythmic, beginning in the neck and arms. The eyes were fixed and staring, and the pupils widely dilated. The convulsions ceased after an hour and a half, but soon began again. Collapse gradually deepened, and death occurred about fifteen hours after seizure. Autopsy showed only a general hyperemia

[1] *Centralbl. f. Bakteriol.*, etc., 1889, vol. vi., p. 289.
[2] *Brit. Med. Jour.*, November 11, 1899.

of the stomach and intestines, a few hemorrhagic erosions of the gastric mucous membrane, and cloudy swelling of the cortex of the kidneys, with a few scattered hemorrhages. Bacteriologic examination of the meat revealed Gärtner's bacillus.

Drs. Wilkinson, Ashton, and Durham[1] have reported a case that involved more than 50 persons who ate of a lot of imperfectly cooked veal-pies. The symptoms began in from five to fourteen hours, and, as a rule, were severe from the start. The chief manifestations were severe and uncontrollable vomiting and diarrhea, accompanied at first by shivering and followed by collapse. The dejecta were at first grass-green, then dark green, and highly offensive. In very few cases they contained blood. The severity of the diarrhea increased on the second day, and in one instance the patient was purged forty or more times in a single day. In some there were violent abdominal pains and in several the abdomen was swollen and tender. Many had severe pains in the back.

In the worst cases the patients became semicomatose, restless, and delirious in the course of a few hours. Occasionally, there were disturbances of vision that lasted until the temperature, which ranged from 100° F. in the mildest to 104.5° F. in the severest cases, became normal. The pulse was very rapid, weak, and dicrotic. In many instances the patient was markedly cyanotic and had some difficulty in breathing. Many had herpes about the lips on the third to the sixth day, and some a rash, followed by desquamation. Some had cramps, and nearly all had muscular pain and stiffness. In the severe cases convalescence was very prolonged, some being still weak after three and a half months. Four of the victims died, and in two cases autopsies were performed. The brain surface showed slight congestion; the small intestine showed congested patches, which became larger and more numerous lower down and did not correspond with Peyer's patches. The whole lower third was highly congested and contained some yellow diarrheic fluid. Beyond these appearances there was nothing noteworthy.

The meat that caused the trouble was from an apparently healthy calf, parts of which were made into pies by other persons whose product caused no disturbance whatever. But the baker to whom the trouble was traced made 160 pies, which were baked in not less than three nor more than five batches, each batch being about twenty minutes in the oven. A study of the whole case led to the conclusion that one whole batch was insufficiently cooked and that the outbreak was due to the presence of Gärtner's *Bacillus enteritidis*. The four fatal cases were due to pies that were two or more days old when eaten, which period must have allowed extensive multiplication of the organisms.

Treatment of Food-Poisoning.—As to treatment of cases of food-poisoning, the cases cited show the impossibility of laying down any general rules. Naturally, one may conclude that the first step to be taken should look, when possible, to the prompt removal of the offending material; but as will have been noted, the onset is often so

[1] *Public Health*, January, 1899, and *Brit. Med. Jour.*, December 17, 1898.

delayed that no other course is open than treatment according to th peculiarities of individual cases. In those cases in which the symp toms point to poisoning of the central nervous system it is obvious tha until the subject shall have been much more extensively studied fro the standpoint of pathologic chemistry, the practitioner must be oblige to grope largely in the dark. Saline infusions, hypodermically or intra venously, may often be used to advantage, as they promote the mor rapid elimination of the offending toxin.

In the study of cases of meat-poisoning it is noteworthy that mut ton and lamb have thus far not been implicated as a cause.

PTOMAINS AND OTHER BACTERIAL PRODUCTS IN THEIR RELATION TO TOXICOLOGY.

THE work of the toxicologist and that of the bacteriologist overlap in many places. In fact it has become quite necessary for the toxicologist to have some accurate knowledge of bacteriologic methods of study. It is the purpose of this section to point out and to dwell to some extent upon those subjects that concern both the bacteriologist and the toxicologist. It has been stated that the chemical tests, especially the color-reactions for certain vegetable alkaloids, may be simulated so closely by certain bacteriologic products that it becomes impossible to distinguish with certainty between them. If this be true, the possibility of mistaking putrefactive substances for vegetable alkaloids should always be borne in mind in making medicolegal investigations. We will now proceed to the discussion of this subject as applied to certain individual alkaloids.

THE TESTS FOR MORPHIN.[1]

In some notable trials for murder, question has been raised concerning the validity of certain color-reactions obtained and supposed to indicate the presence of morphin. In fact it may be stated that toxicologists are now divided in their opinion concerning the nature of the evidence that must be obtained before the presence of morphin in cadaveric tissue can be considered as established. There are those who hold that this alkaloid must be obtained in crystalline form; others believe that certain color-reactions obtained from amorphous and colored residues are sufficient to justify the chemist in swearing to the presence of morphin. Some of the instances in which this question has been raised may be discussed profitably.

In the Songzogna trial at Cremona, Italy, the experts seem to have confounded a basic putrefactive substance with morphin. This substance was not removed from either alkaline or acid solutions with ether, but could be extracted with amylic alcohol. It reduced iodic acid, but in its other reactions, as well as in its physiologic properties, it bore no resemblance to morphin. In frogs it arrested the heart in systole; this does not happen in poisoning with morphin. It failed to give both the ferric chlorid and the Pellagri test for morphin.[2] In the same body there was

[1] Compare pp. 456, 457, 498, 499, 500, and 501 in section on Alkaloidal Poison.

[2] The Pellagri test for morphin: The substance is treated with hydrochloric acid, to which a little sulphuric acid has been added and the mixture moderately heated. After cooling, the residue is mixed first with a drop of hydrochloric acid and then with a slight excess of sodium bicarbonate; a little water is added and afterward a drop or two of a solution of iodin in alcohol, when, in the presence of morphin, a green color is produced.

found a substance that was extracted from alkaline solutions with ether, and that gave, with hydrochloric acid and a few drops of sulphuric acid, on the application of heat, a reddish residue similar to that obtained by the same reagents with codein, but in its other reactions it did not resemble this alkaloid. Selmi clearly demonstrated that the substance obtained from this body was not morphin.

Some years ago the writer, in making a toxicologic examination of a stomach and liver, following the method of Dragendorff, obtained in the amylic alcohol extract from alkaline solution a residue that gave with more or less distinctness all the principal color-tests for morphin; but failing to obtain crystals that could be identified as those of this alkaloid, morphin was not reported. Haines, working with the same material, obtained similar reactions, but he also was unable to secure the crystals and made a negative report. Afterward it was quite positively shown that death had been caused in this case by a blow on the back of the head with a heavy piece of iron.

In the Buchanan case in New York the symptoms as testified to by the attending physician clearly were not incompatible with those that might be due to disease. The chemists for the prosecution swore to the presence of morphin and atropin in the dead body. The tests upon which they relied were the ordinary chemical reactions, and the question arose as to whether or not they were sufficiently distinctive. The writer, in the presence of the opposing experts, made duplicate tests with a solution of morphin and a solution containing putrefactive products. With these all the color-reactions were produced, and the experts for the prosecution decided, after seeing the tests made with the two fluids, that morphin was present in the one that actually contained no morphin.

Indol, skatol, phenol, and derivatives of these bodies are constantly being formed both in the intestines of the living man and in the liver and adjacent tissues for some time after death. Several of these substances give color-reactions that closely resemble those given by morphin. So great is this resemblance that the conscientious chemist will be very careful before he is certain that he has obtained from the tissues in a case of medicolegal inquiry positive evidence of the presence of morphin. I have stated that indol, its allies and derivatives, are present in decomposing tissue, and it should be further stated that the number of indol derivatives is by no means small. Many of these substances give brilliant color-reactions. There has been some difference of opinion as to the identity of indol obtained by putrefaction and that which results from the reduction of indigo. According to Baumann, neither indol nor skatol originates directly from proteids, but both result from the decomposition of a substance that is not proteid in character, and that is soluble in ether containing alcohol. Skatol is methyl-indol. Indoxyl is an easily decomposable substance giving some striking color-reactions, among which may be mentioned the production of indigo-blue with ferric chlorid, in the presence of a trace of free hydrochloric acid. Skatol carbonic acid is another product of putrefaction, Salkowski hav-

ing obtained 1.3 grams of this substance from 2 kilograms of moist fibrin after twenty-six days of putrefaction. Among the known color-reactions of this substance Hoppe-Seyler mentions the following:

1. If a dilute solution of this acid (1 : 1000) be treated with a few drops of pure hydrochloric acid of 1.2 specific gravity, and then with a few drops of potassium nitrate solution (0.2 per cent.), a cherry-red coloration is produced, and, later, a red precipitate falls.

2. If such a solution be mixed with an equal volume of hydrochloric acid and then a few drops of chlorinated lime solution (0.5 per cent.) be added, a purple-red color is produced.

3. Treated with a few drops of hydrochloric acid, and then with two or three drops of a very dilute solution of ferric chlorid and heated, the mixture becomes intensely violet before boiling. It is worthy of note that skatol carbonic acid is non-volatile. Skatol acetic acid has been obtained by Nencki by the anaërobic putrefaction of serum-albumin. The aqueous solutions of this substance give, with ferric chlorid, a white cloudiness that on warming becomes a brick red, and in more concentrated solution, fire red. Both indigo-red and indigo-blue may be formed by the oxidation of indol.

Knowing that indol and its derivatives are formed in anaërobic putrefaction, and that in Dragendorff's scheme for the separation and identification of vegetable alkaloids these substances appear in the residues that are tested for morphin, and knowing the great number and variety of color-reactions given by these substances, it may be asked how much reliance can be placed on the color-tests for morphin.

Besides the indol bodies, certain other substances are formed in the anaërobic putrefaction of proteid substances. Among these are certain aromatic compounds due to the putrefaction of tyrosin. The following may be mentioned:

1. *Parahydrocumaric or Parahydroxyphenyl-propionic Acid.*—This substance gives with ferric chlorid a distinct but evanescent blue coloration.

2. *Parahydroxyphenyl-acetic Acid.*—This substance gives with ferric chlorid a pale greenish violet, which soon changes to a dirty green color.

Among other products of the anaërobic putrefaction of proteids phenol and parakresol may be mentioned. Phenol gives with ferric chlorid a violet color, while parakresol gives with the same reagent a blue coloration.

In order to ascertain how far the color-reactions for morphin might be simulated by the products obtained by the putrefaction of viscera, the writer has made the following experiment:

Five kilograms of ox liver were chopped finely, mixed with two grams of white arsenic dissolved in caustic potash, and placed in a large bottle. The arsenic was added in this experiment because the body of which an analysis had been made in the Buchanan case was embalmed with arsenic directly after death. A glass tube bent at right angles was inserted in the center of the cork of this bottle, while the other end of the tube

was connected by means of a short piece of rubber tubing with a Drechsel wash-bottle. The other arm of the wash-bottle was connected with a receiver filled with water. The rubber connecting the large bottle with the wash-bottle was supplied with a clamp. During the first fifteen or twenty days this clamp was left open, and a large amount of gas passed through the wash-bottle and collected in the receiver. The time during which gas continues to be given off varies according to temperature and other conditions. However, after a while the liberation of gas ceases and the water rises in the receiver, absorbing the collected gas. When this occurred in our experiment, the bottle containing the tissue and the wash-bottle were disconnected, and the clamp on the rubber tube was closed. By this time the chopped liver had become sufficiently fluid to absorb the gas as fast as it was formed, and in case the bottles are not disconnected, the water in the wash-bottle may be drawn back into the larger bottle containing the liver.

The fermentation was allowed to continue for thirty days. Then the contents of the bottle, decidedly acid in reaction, and giving off a rather pleasant ethereal odor, was poured into a large dish. A considerable portion of the tissue had become fluid by this time. One kilogram of this decomposed tissue was placed in each of three evaporating dishes, and these were marked A, B, and C. To B, 130 milligrams of morphin sulphate was added, and to C the same amount of morphin sulphate, together with 0.5 of a gram each of indol, skatol, and phenol. No addition was made to A. These separate portions were carried through all the manipulations recommended by Dragendorff[1] in his processes for the separation and recovery of morphin.

To each 100 c.c. of the fluid 5 c.c. of dilute (1 : 5) sulphuric acid was added. Then 500 c.c. of distilled water was added to each dish, and these were kept at from 40° to 50° C. for eight hours. After this time each portion was filtered through a plaited filter. The fluid passed through quickly, and formed a clear brownish filtrate. The filtrates were evaporated at 50° C. to 600 c.c. each, and four volumes of absolute alcohol were added to each portion. These mixtures were allowed to stand for twelve hours, and in each a brown resinous precipitate formed. After filtration the alcohol was removed by distillation, a fatty residue was found in each flask on the removal of the alcohol, and this was removed by filtration.

The acid solutions were then thoroughly shaken, each with four volumes of petroleum ether. The ethereal layers, when drawn off and evaporated in portions, left slight residues. The residues from A and B gave no reaction on the application of the color-tests for morphin mentioned below. The residue from C showed minute traces of indol with nitric acid alone, and with sulphuric acid containing nitric. The acid solutions were next shaken with benzene. The benzene residues gave no response to the morphin tests in any of the extracts. Chloroform was then applied as a solvent. The residues thus obtained also failed

[1] For description of the Dragendorff process see p. 331 in section on General Principles of Toxicology.

to give morphin reactions. The acid solutions were now rendered alkaline with ammonium hydroxid, and shaken successively with petroleum ether, benzol, and chloroform. None of the residues obtained by these solvents responded to the morphin test.

The alkaline solutions, having been subjected to the above-mentioned process of purification, were shaken, each with five volumes of amylic alcohol. The shaking was frequently repeated during the afternoon, and then the mixtures were placed in separators, and allowed to stand for eighteen hours. The amylic alcohol extracts from all three portions evaporated on the water-bath gave the following reactions :[1]

REAGENTS.	A, B, C.
Nitric acid.	All gave a lemon-brown color.
Sulphuric acid.	None showed any change.
Sulphuric with nitric acid.	All gave a lemon-yellow, slowly changing to a pink.
Ferric chlorid.	All gave a dirty green.
Iodic acid.	All promptly reduced the iodic acid.
Fröhde's reagent.	All gave a blue color without any violet.
Sulphuric acid and cane-sugar.	All became brownish red, changing to a wine red.

Portions of the amylic alcohol extracts allowed to evaporate spontaneously showed the same reactions as those given above.

The remaining portions of the amylic alcohol solutions were now shaken with distilled water acidified with sulphuric acid. After separation, portions of the amylic alcohol were evaporated and subjected to the above-mentioned tests, with negative results in each case. This shows that amylic alcohol does not dissolve from acid solutions the substance or substances interfering with the morphin test.

The acid aqueous solutions of A, B, and C were again rendered alkaline with ammonium hydroxid and shaken with amylic alcohol. The residues from these amylic alcohol extracts were evaporated and subjected to the following tests :

REAGENTS.	A, B, C.
Nitric acid.	All became lemon-yellow.
Sulphuric acid.	No change in any.
Sulphuric with nitric.	All became lemon-yellow.
Ferric chlorid.	All became bluish green.
Iodic acid.	All promptly reduced iodic acid.
Fröhde's reagent.	All became blue with a faint and evanescent purple in B and C.
Pellagri's test.	All responded promptly.

The above-mentioned experiments convinced the writer that the tests for morphin, by following the scheme of Dragendorff, are not trustworthy. Naturally the question arises, what is the nature of the substance or substances that give these color-reactions. It has already been hinted that indol with its allies and derivatives is accountable for many of these reactions. The probabilities in favor of this may be briefly stated as follows :

[1] Compare Chemical Tests for Morphin, p. 497 *et seq.*, in section on Alkaloidal Poisons.

1. Germs that produce indol and its derivatives are constant representatives of the bacterial flora of the upper portion of the small intestine. There are many indol-forming germs, and while some of these may be present in any tissue, they are certainly present in health and in disease, during life and after death, in both the small and the large intestine of man and other animals.

2. Indol and its derivatives are products of anaërobic putrefaction, and this accounts for the fact that the reactions referred to above are not familiar to those toxicologists who have experimented with tissues allowed to putrefy in the presence of oxygen.

It was from a belief that indol and its derivatives had been in some instances mistaken for morphin that the writer was led to add these substances to one portion of tissue in the experiments mentioned above.

Assisted by Dr. Houghton, the writer made some further experiments with indol obtained from different sources. The samples of indol used in these experiments may be grouped as follows:

No. 1. This was prepared some years ago by the writer from decomposing pancreatic tissue. It is a brown, granular substance, and is probably not chemically pure. This fact, however, does not unfit this sample for experiments on the point under consideration, because any impurities that it may contain originated in the decomposing tissue, and may be present in the same substance obtained from like tissue.

No. 2, obtained from Merck. The order was simply for "indol," without any specifications whether it should be synthetic or putrefactive. It is brownish red in color.

No. 3, obtained from Schuchardt, and ordered as synthetic indol, which it undoubtedly is. This sample is white and in flakes.

No. 4, obtained from Kahlbaum. Putrefactive indol was ordered, and the label is simply "indol"; this sample consists of white flakes. These samples were submitted to the following tests:

Reagent.	No. 1.	No. 2.	No. 3.	No. 4.	No. 5. Pure morphin sulphate.
Nitric acid.	Bluish black with violet border.	Reddish brown.	Reddish brown.	Reddish brown.	Brownish red, passing into lemon-yellow.
Sulphuric acid.	Yellowish green.	Brown.	Greenish yellow.	Brownish red.	Faint yellow.
Sulphuric acid with nitric acid	Same as with nitric acid alone.	Same as with nitric acid alone.	Same as with nitric acid alone.	Same as with nitric acid alone.	Brownish red.
Ferric chlorid.	No change at first, then became greenish blue.	No change at first, then became greenish blue.	No change at first, then became greenish blue.	No change at first, then became greenish blue.	Blue.
Iodic acid.	No reaction.	No reaction.	No reaction.	No reaction.	Reduced
Frohde's reagent.	Reddish, then dark blue.	Reddish, then greenish blue.	Reddish, then greenish blue.	Reddish, then greenish blue.	Purple, then blue.

While it would be perfectly easy to distinguish pure morphin from either indol or skatol or any of their derivatives, it must be admitted, from the results of the experiments already detailed, that the separation of morphin from decomposing tissue, and its identification by the methods

now generally employed, are so uncertain that no great reliance can be given them.

Another noted case in which the tests for morphin have been called into question is that of Urbino de Freitas, which occurred in Portugal in 1893. The experts for the prosecution reported the presence of morphin, narcein, and delphinin in the tissues. The experts for the defense offered the following criticisms:

(1) That the chemical examination of the viscera was made at a time when putrefaction was well advanced. (2) That amylic alcohol was used in extracting the alkaloids, and, moreover, that the amylic alcohol was not pure; that it contained many basic substances, and possibly furfurol. (3) That the method used by the prosecution in detecting the presence of morphin and narcein in the urine was defective. (4) That the physiologic results obtained with the syrupy extracts did not show the presence of vegetable poison. (5) That the color-reactions obtained for the alkaloids mentioned were not sufficient to enable one to make a positive statement concerning their presence.

The controversy between the experts on the two sides of this case finally involved most of the prominent toxicologists of Europe, inasmuch as the evidence was submitted and testimony obtained from these.

As in the Buchanan trial, much stress was given to the employment of amylic alcohol in the Portuguese case. In regard to this solvent, Beckurts affirmed that it is the best solvent possible for putrefactive substances. He adds that it is difficult to remove all traces of amylic alcohol from its residues; that these residues give many color-reactions, and that the employment of these residues in physiologic tests is likely to lead to error.

Bischoff and Brieger state that the use of amylic alcohol as a solvent in toxicologic work is likely to lead the analyst into error. Furthermore, they claim that amylic alcohol gives residues that are purified with great difficulty. In conclusion on this point they recommend that amylic alcohol should not be employed in toxicologic research.

The above-mentioned German experts furthermore state that the impurities in amylic alcohol partially at least explain the color-reactions and the results obtained in the physiologic experiments. They think that these impurities, together with certain other substances extracted by the amylic alcohol, led the Portuguese chemists into the erroneous conclusion that they had obtained evidence of the presence of morphin, narcein, and delphinin. The Portuguese experts replied that the impurities said to be in the amylic alcohol were imaginary, and they think that they proved that the amylic alcohol used by them did not contain any impurities, by evaporating some of this alcohol and studying some of the residue thus obtained. However, this proof does not amount to much because the impurities in amylic alcohol when mixed with residues obtained by the extraction of organic material will give very different color-reactions from the residue obtained by the evaporation of the amylic alcohol alone. It was asserted by the German experts that the re-

actions obtained by the Portuguese chemists with Fröhde's reagent and with ferric chlorid might be due to pyridin bases. The Portuguese experts answered this by stating that neither of these reagents gives any color with pyridin. This again shows their lack of knowledge on the subject because they could not possibly have experimented with any large number of pyridin bases, the exact nature of many of which is still unknown.

The Portuguese experts claimed to have extracted morphin and narcein from urine by first shaking the acid urine with amylic alcohol in order to remove urea and bile acids, and then rendering the urine ammoniacal and again extracting with amylic alcohol. The German experts criticized this method and state that in this way substances might be obtained from normal urine that would give some of the color-reactions supposed to be indicative of the presence of morphin. In regard to the results obtained by injecting the amylic alcohol extract into frogs Beckurts states that such a residue might contain potash, ammonia, pyridin bases, and ptomains, and that the results obtained in the experiments with the frogs might have been due to one or more of these substances.

The Portuguese experts relied for their tests for narcein upon the formation of a blue precipitate with the double iodid of potassium and zinc. In regard to this test the German experts state that with 0.1 of a milligram of pure narcein this test might be of importance, but when applied to an impure extract, the test is without value.

The Portuguese experts relied for their evidence as to the presence of morphin upon the iodic acid reaction, the test with Fröhde's reagent, the test with Lafon's reagent,[1] and the evidence of alkaloids in the residue obtained by the use of the general alkaloidal reagents. The German experts state that the reaction for morphin with ferric chlorid might be due to small quantities of pyrocatechin or kresol or homogentisinic acid, all of which substances may be found in normal urine. They also state that certain substances in normal urine may give the morphin reaction, or one very similar to it, with iodic acid and with Fröhde's reagent. In regard to the iodic acid test, Beckurts states that this reaction may be obtained in every toxicologic examination. Furthermore he states that the color-reaction with Fröhde's reagent is not characteristic, and that it may be produced by many glucosids and by certain coloring substances.

In regard to the test for delphinin, Bischoff and Brieger state that this alkaloid cannot be obtained by Dragendorff's method, inasmuch as it would be destroyed by the mineral acids and other reagents used in this method.

The Portuguese chemists claim that they obtained 200 milligrams of vegetable alkaloid by the method employed. The opposing experts state that if this large amount of alkaloid had been obtained, the Portuguese experts should have exhibited some of the alkaloid in the trial of

[1] Lafon's reagent: A solution of 1 gram of ammonium selenite in 20 c.c. of strong sulphuric acid.

the case, inasmuch as this amount would have been more than they need to have used in their tests. The answer to this is that other chemists have also obtained large amounts of vegetable alkaloids in toxicologic cases. Ponchet claimed to have isolated 148 milligrams of strychnin from 510 grams of viscera in a medicolegal case. Rouan states that he isolated 106 milligrams of the same alkaloid from 1920 grams of tissue. Stas, in the celebrated Bocarmé case, reported the finding of an enormous amount of nicotin. In one case Dragendorff reported that he obtained more than ½ gram of morphin from 420 grams of tissue.

The question whether or not a chemist should be expected to obtain any of the alkaloids in medicolegal cases in sufficient quantity to enable him to exhibit the poison in court is an interesting one. The writer is inclined to think that this is not in all cases possible. However, when, as in the case under discussion, the amount is large, the chemist, in order to protect himself, should be able to exhibit the poison in court. No one questions the desirability of offering such evidence in cases of poisoning with metallic substances, but when a vegetable alkaloid has been used, the difficulty of obtaining the alkaloid in a state of perfect purity, and the small amount of the poison usually obtained, render the requirement of such an exhibition questionable.

In a review of the above-mentioned case Husemann thinks it quite certain that the Portuguese experts mistook putrefactive products for vegetable alkaloids. Dragendorff, whose method the Portuguese experts employed, agreed with their opponents, and gave it as his opinion that putrefactive substances had been mistaken for vegetable alkaloids. Stephenson, of London, and Lewin, of Berlin, came to the same conclusion.

I have presented the medicolegal testimony concerning this Portuguese case in some detail because I have long believed that the methods now relied upon for the detection of morphin in medicolegal cases is not trustworthy. It is an interesting fact that the criticisms made by the writer upon the experts for the prosecution in the Buchanan case are almost identical with those made by the distinguished toxicologists of Europe in the Portuguese case.

CONIIN.

In quite a number of trials for murder the evidence concerning the presence of coniin in the tissues has been brought into question. One of the most celebrated of these is known as the Brandes-Krebs case, which was tried in Braunschweig in 1874. From the tissues submitted to them two chemists obtained, in addition to arsenic, an alkaloidal substance that they pronounced coniin. The distinguished toxicologist, Otto, was called upon to make a further examination of this substance. He reported that it was neither coniin nor nicotin, nor any other vegetable alkaloid with which he was acquainted. He converted it into an oxalate, dissolved it in alcohol, evaporated the alcohol, dissolved the residue in water, rendered this solution alkaline with potassium hydroxid,

and extracted the base with petroleum ether. On evaporation of this solvent the substance appeared as a bright-yellow oil that had a strong, unpleasant odor, differing, however, quite markedly from that of coniin. It was strongly alkaline, and had an intensely bitter taste. At ordinary temperature it was volatile. From its aqueous solutions it was precipitated by the chlorids of gold, platinum, and mercury. In these reactions it resembled nicotin, but it differed wholly from this alkaloid in the double refractive and crystalline character of its hydrochlorid. With an ethereal solution of iodin this substance did not give the Roussin test for nicotin, but, instead of the long, ruby-red crystals, there appeared small, dark-green, needle-shaped crystals.

This unknown substance was found to be markedly poisonous. Seven centigrams (1 grain) injected subcutaneously into a large frog produced instantaneous death, and 44 milligrams (0.7 grain) given to a pigeon caused a similar result. On account of its poisonous properties the jury of medical experts decided that the substance was a vegetable alkaloid. It is needless to say that this decision is not in accord with what all now know concerning the action of many of the basic products of putrefaction.

Brouardel and Boutmy obtained from the body of a woman, who had died after suffering, with ten other persons, from choleraic symptoms following the eating of a stuffed goose, a base that gave the odor of coniin and the same reactions as this base with gold chlorid and with iodin in potassium iodid. The same substance was found in parts of the goose not eaten. It differed from coniin inasmuch as it did not give the red coloration with strong hydrochloric acid, and did not form butyric acid on oxidation. It was poisonous, but in its physiologic action it did not resemble coniin.

Selmi repeatedly found in decomposing animal tissue substances resembling coniin. In one instance he made an alcoholic extract of the tissue and distilled the solution thus obtained. The distillate was acidified with hydrochloric acid and evaporated; the residue was treated with barium hydroxid and ether, and then the ether was allowed to evaporate spontaneously; he obtained a residue of volatile bases, the greater part of which consisted of trimethylamin. After removing the trimethylamin the residue had the odor of the urine of the mouse. It should be understood that in this case there was no possibility of coniin being present in the tissue. In another instance Selmi obtained an unmistakable coniin odor from a chloroform extract of the viscera of a person who had been buried for six months; and in still another instance he obtained a like substance from the tissue of a person who had been buried for ten months. Two or three drops of an aqueous solution of the alkaline residue of the chloroform extract allowed to evaporate on a piece of glass gave off so penetrating an odor that Selmi was compelled to withdraw from close proximity to the substance. The odor imparted to his hands in testing the solutions remained for half an hour. This volatile base was, in the opinion of Selmi, formed by the spontaneous decomposition of other ptomains.

An aqueous solution of a putrefactive base obtained by Selmi by extraction with ether, according to the Stas-Otto method,[1] from the undecomposed parts of a cadaver, had no marked odor, but after having been kept for a long time in a sealed tube, and doubtlessly having undergone partial decomposition, it not only gave off a distinctly coniin odor, but the vapor turned red litmus-paper blue. Again, the sulphate of a basic substance obtained by Selmi from putrid egg-albumen on standing formed in two layers, one of which was a golden-yellow liquid that, on being treated with barium hydroxid, gave off ammonia, and later the odor of coniin. Since butyric and acetic acids were formed by the oxidation of this base, Selmi concluded that he had found either coniin or methylconiin, and that it was formed by the oxidation of certain non-volatile ptomains or by the action of amido-bases on volatile fatty acids. From this it will be seen that Selmi believed in the origin of coniin or closely allied bases in putrid matter; or, as it may be stated, in the existence of a cadaveric coniin.

In a trial in East Prussia Sonnenschein has reported upon a substance that had been pronounced the alkaloid of the water hemlock (*Cicuta virosa*). Otto and Husemann believe this substance to be cadaveric coniin. Otto states that the symptoms reported in the case were not those of either coniin or cicuta. This substance was obtained from the tissue six weeks after the exhumation of the body, which had been buried for three months. This base had the odor of coniin, the taste of tobacco, gave with potassium bichromate and sulphuric acid the odor of butyric acid, and behaved with reagents like coniin.

Husemann states—and the present writer certainly agrees with him—that at present it is very difficult, if not impossible, for the chemist to state with certainty that he has detected true coniin in the dead body. The symptoms and the postmortem appearances must conform most closely with those induced by the vegetable alkaloid. The analysis must be made before decomposition sets in, and the amount of the base found must be sufficient for physiologic experiments to be made with it.

NICOTIN.

From the decomposed intestines of a man who had been dead six weeks Wolckenhaar obtained, by extraction with ether from an alkaline solution, a base that bore a close resemblance to nicotin. This substance was fluid and at first yellow in color, but on being exposed to the air it became brownish yellow. It was strongly alkaline in reaction, and gave off an odor resembling nicotin, but stronger, not ethereal, benumbing, and similar to that of fresh poppy-heads. It was freely soluble in water, and its aqueous solutions did not become cloudy on the application of heat. They were bitter and pungent to the taste. The peculiar odor did not disappear on saturating the base with oxalic acid. The hydrochlorid was yellow, had a strong odor, and became moist on ex-

[1] For description of the Stas-Otto method see p. 430 in section on General Principles of Toxicology.

posure to the air. Under the microscope this compound showed no crystalline structure, differing in this respect from the hydrochlorid of nicotin. Furthermore, it differed from nicotin in its reactions with potassio-bismuthic iodid, gold chlorid, iodin solution, mercuric chlorid, and platinum chlorid. It also failed to give the Roussin test for nicotin. Again, it could not be identified with trimethylamin, spartein, mercurialin (methylamin), lobelin, or other non-fluid and volatile bases.

Rörsch, Fassbender, Schwanert, Liebermann, and Selmi, as well as others, have found nicotin-like substances in decomposing animal tissue when there was no possibility of true nicotin being present.

STRYCHNIN.

At a trial for murder at Verona, Italy, Ciotta obtained from the exhumed but only slightly decomposed body an alkaloid that gave a crystalline precipitate with iodin and hydriodic acid, a red coloration with hydriodic acid, and a color-test similar to that of strychnin with sulphuric acid and potassium bichromate and with other oxidizing agents. This substance was strongly poisonous, but in its action it differed materially from strychnin, inasmuch as it did not produce the tetanic convulsions that are so characteristic of this vegetable alkaloid. Ciotta was in doubt concerning the exact nature of this body, but pronounced it probably identical with strychnin. The tissue was submitted to Selmi for further investigation and for his opinion. Selmi ascertained that the substance that gave the color-reaction was not crystalline, and that there was only the presumption of a bitter taste to it, while 1 part of strychnin to 40,000 parts of water is intensely bitter. Selmi furthermore convinced himself that many putrefactive substances give reactions similar to strychnin with iodin in hydriodic acid, and with hydriodic acid alone. He also held that the physiologic properties of this substance were such that it could not be strychnin. What it was he was not able to determine. It could hardly have been quebrachin, which gives a reaction with sulphuric acid and potassium bichromate similar to that of strychnin, because quebracho bark, in which this alkaloid is found, was not at that time used as a medicine or known in Italy.

Ptomains giving reactions similar to those of strychnin and also causing tetanic symptoms have been found in Italy in decomposed cornmeal. Selmi studied one of these substances and found that it differed from strychnin inasmuch as it could not be extracted with ether. Lombroso has named the poisonous substance found in decomposed cornmeal pellagrocein, but this is really a mixture of putrefactive substances, some of which cause narcosis and paralysis, while others produce the symptoms of nicotin poisoning instead of the spasms caused by strychnin.

In cases of strychnin poisoning the toxicologist should always be able to obtain this substance in crystalline form. Furthermore, he should never neglect the physiologic test, notwithstanding the fact that the chemical reactions for strychnin are more characteristic than for most

vegetable alkaloids. The most positive evidence furnished by these tests should always be supplemented by the physiologic test. The writer has personal knowledge of a case in which there can be little doubt that strychnin was present, but the prosecution failed to convict because the toxicologist had not protected himself against the possibility of error by supplementing the chemical reactions by the physiologic test.

ATROPIN.

Among the products of putrefaction of animal tissue there will frequently be found substances which, in their mydriatic properties, resemble atropin and hyoscyamin. Every one who has worked extensively with decomposing animal tissue has met with bodies of this kind, and more than once they have figured in medicolegal cases. To this class belongs the substance observed by Zuelzer and Sonnenschein. This body was obtained from alkaline solution by extraction with ether. It formed microscopic crystals, an aqueous solution of which, when applied to the conjunctiva, produced a mydriatic effect, and when administered internally, increased the action of the heart and arrested the peristaltic movements of the intestines. Further than this, with certain alkaloidal reagents, such as platinum chlorid, it resembled atropin. However, when heated with sulphuric acid and oxidizing reagents, it failed to give the odor of blossoms (Reuss's test). However, Selmi found ptomatropins that, with sulphuric acid and oxidizing agents, did give the blossom odor as distinctly as vegetable atropin does. These putrefactive bases also developed this odor spontaneously after standing for two or three days, and this does not happen with atropin. The odor was also produced with the ptomatropins by nitric and sulphuric acids, both in the cold and on the application of heat, while these acids in the cold do not produce this odor with true atropin.

Substances belonging to the group of ptomatropins have been found in decomposing fish, canned beef, putrid game, and poisonous sausage. We cannot positively say whether there is only one or more of these ptomatropins. Some of them, at least, are poisonous. The symptoms often closely resemble those of belladonna poisoning. The throat becomes dry, the muscles of deglutition seem to be paralyzed, the secretion of perspiration and saliva is arrested, mydriasis may be marked, and there may be paralysis of accommodation, ptosis, and strabismus. In some instances delirium resembling that of belladonna is marked, while in other cases convulsions appear. The effect upon the heart is to increase the number of beats and decrease the strength of the beat. The tongue is often heavily coated, and in the most dangerous of these cases constipation is most obstinate. The victim becomes exceedingly weak, and in some instances the voice may be wholly lost. In postmortem examination after poisoning with food containing one of these putrefactive ptomatropins the pharynx is found to be swollen, hemorrhagic spots are observed in the esophagus, stomach, and intestines, the solitary follicles show cloudy swelling, and marked degeneration in the

By digesting this substance with gastric juice and again extracting he obtained a body that behaved with sulphuric acid similar to curarin.

DELPHININ.

This substance first attracted the attention to the possibility of mistaking putrefactive products for vegetable poisons. In 1870 General Gibbone, an Italian of prominence, died suddenly at Rome. His servant was accused of having poisoned him. Two chemists of reputation reported the presence of delphinin in the viscera. It seemed somewhat improbable that the servant should know anything of so rare a substance or that he should have been able to obtain it. However, two or more varieties of staphisagria grow in southern Italy, and it was barely possible that the servant had used some preparation made by himself from the plant. The supposed alkaloid was given to Selmi, of Bologna, for further study. It was extracted from alkaline solutions by ether. When heated with phosphoric acid it became red, and when brought in contact with concentrated sulphuric acid, it turned reddish brown. In these tests this substance resembled delphinin, but with sulphuric acid and bromin water, also with Fröhde's reagent, the colorations characteristic of the vegetable alkaloid failed to appear. Furthermore, Selmi stated that delphinin gives the following reactions, to which the suspected substance did not respond: (1) Delphinin dissolved in ether and treated with a freshly prepared ethereal solution of platinum chlorid gives a white, flocculent precipitate that is insoluble in an equal volume of absolute alcohol. (2) Delphinin gives precipitates with thiosulphate of sodium and gold, and with a sulphuric acid solution of the thiosulphate of copper and sodium, the latter precipitate being soluble in an excess of the reagent. Finally, Ciaccia and Vella showed that while delphinin arrests the heart of the frog in diastole, the suspected substance arrested it in systole. Thus it was most conclusively demonstrated that the suspected substance was not delphinin.

COLCHICIN.

In a few medicolegal cases the tests for this substance have been brought into question. In a case of suspected poisoning Baumert examined the tissues twenty-two months after death. He found a substance that gave many of the reactions of colchicin. It was extracted from acid solutions with ether, to which it imparted a yellow color. On evaporation of the ether a yellow, amorphous substance remained, and this, dissolved in warm water, again imparted a yellow coloration. It could also be extracted from acid solution by chloroform, benzene, and amylic alcohol, but not by petroleum ether. It was not so easily removed from alkaline solutions. It was noticed that it imparted to all solvents a yellow color. On evaporation there remained a feebly alkaline residue having a markedly bitter, sharp taste and devoid of crystalline form. This residue dissolved incompletely in water and dilute acid, forming a resin. When this resin was dissolved in dilute sodium

hydroxid and the solution rendered acid by sulphuric acid, the same reactions were obtained as with the original extract. With phosphomolybdic acid, phosphotungstic acid, potassium bismuth iodid, potassium mercuric iodid, iodin in potassium iodid, tannic acid, and gold chlorid this substance gave the same reactions that were obtained by parallel experiments with genuine colchicin; thus the tannic acid precipitates were both soluble in alcohol, and the precipitates with phosphomolybdic acid in both cases became blue on the addition of ammonium hydroxid. It might be remarked here, parenthetically, that this color-reaction cannot serve for the identification of any substance because it is so frequently given in the presence of decomposing matter. Concentrated sulphuric and dilute nitric and hydrochloric acids dissolved the supposed colchicin, forming yellow solutions. Strong nitric acid colored the substance dirty red, scarcely to be called a violet. When this body was purified as much as possible, the coloration with strong nitric acid became a beautiful carmin-red. The addition of water changed the red into yellow, and caustic soda produced a dark, dirty orange. In general, so far as the above-mentioned reactions go, the putrefactive substance agreed with the real colchicin, but the former gave precipitates with picric acid and platinum chlorid, while the latter gives no precipitates with these reagents.

In 1886 Zeisel proposed the following test for colchicin: When a hydrochloric acid solution of colchicin is boiled with ferric chlorid it becomes green, sometimes dark green and cloudy. Now if the fluid be agitated with chloroform, the chloroform will sink, taking with it the coloring-matter, and appearing brownish granite-red or dark, while the supernatant fluid clears up without becoming wholly colorless. This test was applied by Baumert to his putrefactive substance. To from 2 to 5 c.c. of the suspected solution in a test-tube he added from 5 to 10 drops of strong hydrochloric acid, and from 4 to 6 drops of a 10 per cent. solution of ferric chlorid; then heated the mixture directly with a small flame until it was evaporated to half its volume or less. In the presence of 1 milligram of colchicin the originally bright-yellow solution became gradually olive green, and on further condensation dark green and cloudy. On shaking the fluid with chloroform, admitting as much air as possible, the chloroform subsided, having a ruby-red color if as much as 2 milligrams of colchicin were present, and a bright yellow with only 1 milligram, and the supernatant fluid became a beautiful olive green. When ether, petroleum ether, benzene, carbon disulphid, or amylic alcohol was substituted for the chloroform, the coloration did not appear. From this Baumert infers that the red coloring-matter is either soluble in chloroform only, or that it is not formed until chloroform is added. He found this test of great value in deciding as to the identity of the putrefactive body with colchicin. The putrefactive substance did not respond to this test. Later, Brieger examined this putrefactive body and found it to be a peptone-like substance, also that it was wholly inert physiologically.

This substance had previously been studied by Liebermann, who

had also obtained it from a cadaver. His description of this body differs from that of Baumert only in respect to the taste of the substance. Liebermann failed to observe any marked taste in his extract, while, as has been stated, Baumert found a distinctly bitter taste. A colchicin-like substance has been found in beer, and it has been suggested that it was this that the above-mentioned toxicologists found in the bodies examined, but Liebermann states that the man whose body he examined had been a total abstainer from beer.

SOME PROPOSED METHODS OF SEPARATING VEGETABLE ALKALOIDS FROM PUTREFACTIVE PRODUCTS.

The difficulties detailed in the preceding pages have led to numerous attempts to devise methods by means of which vegetable alkaloids may be separated from putrefactive bodies and positively identified. Among the schemes presented for this purpose that devised by Kippenberger is the only one, in the writer's opinion, worthy of mention. (For description of the process see p. 338 in section on General Principles of Toxicology.) This method excludes amylic alcohol from the list of solvents employed, and in this particular at least is commendable.

On some points the author is quite indefinite in his directions, and leaves much to the judgment of the one who attempts to follow his method. In the writer's laboratory a 10 per cent. solution of tannic acid in glycerin has been employed, and the temperature recommended for the coagulation of albuminous bodies has been raised to between 60° and 70° C., and maintained at this point for two hours. The writer can state, from his own experience with this method, that while it has certain advantages over others, it does not wholly solve the difficulty of separating morphin from putrefactive bodies that respond to some of the morphin tests.[1]

[1] Compare p. 457 in section on Alkaloidal Poisons.

THE POSTMORTEM IMBIBITION OF POISONS.

WITHIN recent years the question of the diffusion of poisons through the dead body has acquired importance largely on account of the fact that arsenic, corrosive sublimate, and other poisonous substances are used for the purpose of embalming bodies. A criminal may easily mask his crime if he can find opportunity to introduce, or to have some one else introduce, the poison used in committing the murder into the body after death. On the other hand, a poison may be introduced either accidentally or intentionally into the body after death, and later some one may be accused of having administered that poison during life. The body is then disinterred, the chemist makes his examination, the poison is found in various organs, and the question arises as to whether it was introduced during life or after death. This subject is of sufficient importance to justify us in going somewhat into detail in the study of the experiments that have been made, for the purpose of ascertaining the truth concerning it.

In the first edition of his great work upon toxicology, published in 1845, Orfila speaks as follows on this subject: "It is true that if corrosive sublimate, arsenic, the salts of copper, sulphuric and nitric acid, etc., be introduced into the digestive canal some minutes after death, they induce lesions of tissue resembling up to a certain point those which are developed by the ingestion of these same substances during life. However, it is easy to distinguish these lesions by the following characteristics: (*a*) In case the poison be introduced after death in solid form, it will be found to exist in large quantity only a short distance beyond the point of its application. Those parts of the digestive canal distant from the point of application, depending more or less upon the length of time intervening between the application and the time of examination, will remain unaltered. While, on the other hand, if it be administered during life, and be not expelled by vomiting or otherwise, it will be widely diffused. (*b*) If the poison be introduced in solution after death, it will penetrate to a greater distance, but again there will be a notable difference in the extent of its diffusion from that which occurs after its administration during life. (*c*) When the poison is injected after death, there will be a sharp line of demarcation between the tissues penetrated by it and those to which it has not extended. When an irritant poison is introduced during life, it causes an inflammation of more or less intensity, which insensibly extends into the adjacent tissue, and there is never a sharp line of demarcation. (*d*) The redness, the inflammation, the ulceration, and the other lesions are infinitely greater when the poison is introduced during life than when it is

applied after death. If, on examination of the cadaver, one finds the rectum or the stomach filled with a large quantity of the poison and the inflammation very slight, one can presume that the poison has been applied after death. (*e*) There are certain poisons, such as corrosive sublimate and nitric acid, which cause characteristic lesions when injected immediately after death, and it is impossible positively to tell whether these lesions were caused by administration during life, or by application after death. (*f*) The corrosive poisons, if introduced into the digestive canal twenty-four hours after death, do not develop much redness or inflammation, because they do not extend into the capillaries. (*g*) These poisons may cause lesions which simulate a mild congestion when they are applied one or two hours after death.

"In all cases of this kind it should not be forgotten that imbibition after death takes place very slowly and will be most marked in parts that are below the point of application. In one case in which the sulphate of copper was introduced into the stomach after death, this salt was found to have penetrated the left side of the diaphragm and the left lung, while the right side of the diaphragm and the right lung did not contain the poison. It will also be found in these cases that in postmortem imbibition the poison does not penetrate to the center of certain organs, or, at least, will be found in greater quantity near the surface. If, however, the examination be not made until after many months and after a time when putrefaction has markedly progressed, it is quite difficult to determine whether the poison was introduced before or after death. In such a case much reliance must be placed upon the symptoms and other evidence of poisoning."

In 1850 Kidd published, in the *Dublin Quarterly Journal of Medical Sciences*, some experiments upon cadaveric imbibition. He injected from 4 to 8 ounces of a solution of arsenic containing 10 grains to the ounce into the bodies of a cat and a rabbit, and found, after about a month, that the poison was widely distributed through the adjacent organs. In the rabbit he found that in this way the poison had penetrated as far as the heart and the tissues of the forelegs.

Kidd's work was so well done and is so applicable to similar cases that have arisen in the last few years that we may with profit quote quite extensively from his communication. He summarizes the evidence in the case as follows:

"Mr. Bleazby, a gentleman of independent fortune, died after an illness of short duration, on the sixteenth of October, 1849. After his death rumors arose that he had been poisoned and an inquest was called for. The body was raised on the fifteenth of December, 1849, and the stomach, liver, and kidneys were removed for examination. The body presented appearances which caused the suspicion that a stomach-pump had been introduced after death, and at the inquest a defense was attempted on the ground that the person had died from natural causes, and that poison had been injected into the stomach afterward." The chemist obtained arsenic from the stomach, and unequivocal evidence of its presence in the liver and kidney. The report of the chemist read as

follows : "From the appearance of arsenic in the stomach, kidney, and liver, deponent has no hesitation in stating that in his opinion the death of the individual from whose body these parts were extracted was caused by arsenic. As a matter of opinion, the arsenic must have been received in the stomach before death. It is possible to inject arsenic into the stomach after death, but in that case it could not enter the liver or kidneys ; it is only by absorption that it could reach these organs, and absorption ceases immediately after death."

Kidd made not only the experiments already referred to, but, by means of endosmometers, he showed that a solution of arsenic separated from blood-serum by an animal membrane would diffuse until the two fluids were of practically the same composition. Had Kidd's work been better known, the controversy concerning the diffusion of poisons through the dead body could not have been so sharply contested as it was a few years ago.

In 1852, in a new edition of his work upon toxicology, Orfila reported experiments upon dogs and men. He introduced into the body, through the stomach or rectum, from 30 to 45 grains of arsenic dissolved in a pint of water. The parts of the liver and other organs that lie in contact with the digestive canal were found to contain arsenic, while the other parts were found free from this substance. When the arsenic was introduced into the stomach and the body placed upon the back, the poison was found in the left half of the diaphragm and in the lower lobe of the left lung, while none could be found in the other portions of the diaphragm or in the right lung.

In 1856 three Italians, Moltedo, Ageno, and Granara, published some experiments, of which the following is an abstract : Ten centigrams of arsenious acid was introduced into the empty stomach of a dead rabbit. Twenty-nine hours later, analysis showed the presence of arsenic in the lungs, in the walls of the heart, and in the blood. Twenty centigrams was introduced under like conditions into another animal. Seventeen hours later marked arsenical mirrors were obtained from the blood, the lungs, and the heart, and a smaller mirror from the liver. Similar results were obtained in experiments upon another animal into which 7 centigrams of arsenic was introduced twenty-four hours after death, and the analysis was made twenty-four hours later. In the fourth experiment 15 centigrams of arsenic was introduced into the large intestine, and twenty hours later the poison was found in the kidneys and in the urine. In the fifth experiment 30 centigrams was introduced into the stomach of a fetus. After twenty-four hours marked traces of the poison were found in the kidneys. In a sixth case one-half gram of arsenic was introduced into the stomach of a dead boy. Analysis, made a few hours later, failed to show any evidence of diffusion. Finally, a human liver, with the blood-vessels and gall-ducts tied, was allowed to lie for forty hours in an arsenical solution. The examination of a small piece from the center of the liver failed to show the presence of arsenic. They concluded from their experiments that while arsenic may penetrate the blood, lungs, and heart of a dead body, its presence

in the interior of the liver is positive proof of its absorption during life.

In 1862 Walther reported an interesting case: A woman administered a large quantity of arsenic to another woman, and, observing that the action of the poison was slow, pushed her victim into the river, where she drowned. Postmortem examination, followed by chemical analysis, showed the presence of arsenic in the stomach, the spleen, and the liver, but none in the brain. The question arose on the trial as to whether death was due to the poison or to drowning. In order to solve this question Walther introduced into the stomach of each of three rabbits 2 grains of arsenic. Two of these animals were kept submerged in cold water for four and one-half days, while the third one was allowed to lie in the air. In the submerged animals no arsenic could be found in the liver or spleen, while arsenic was found in both of these organs in the third rabbit. Walther draws the following conclusions from these experiments: (1) Arsenic introduced into the living body is immediately absorbed, the process of absorption going on more or less rapidly according to conditions. (2) The absorption of arsenic stops immediately with death, but begins again with putrefaction, and its diffusion at that time is due to the formation of the arsenid of hydrogen. (3) When one finds arsenic in the liver or spleen of an undecomposed body, it must be assumed that the poison reached these organs during life. (4) When arsenic is found diffused through the tissues of a decomposed body, it may have reached the various tissues either by absorption during life or as a result of putrefaction after death.

In 1882 the question of the postmortem imbibition of arsenic became the turning-point in a trial for murder in Michigan. The facts of the case, so far as expert testimony was concerned, are briefly as follows: Matthew Millard was accused of poisoning his wife with arsenic. The woman was taken sick on April 18, 1882. She was seen nearly every day, and sometimes twice a day, by a physician, and twice the attending physician had counsel. The woman had long been subject to uterine trouble; the nature of this trouble does not seem to have been understood by the attending physician. During her illness she vomited frequently, and, indeed, seldom retained either food or medicine. The testimony as to the symptoms manifested was so confused and conflicting that nothing definite could be made out of it. The attending physician thought that she had fever, but he never took her temperature. Death occurred on the 7th of May. After her death the husband requested the undertaker to embalm her body so as to preserve it until a casket could be brought from Detroit. The undertaker replied that he did not know how to embalm a body. The husband, who had once been an undertaker, requested the man in charge of the remains to obtain arsenic, saying that he (the husband) would embalm the body. On this point there was some conflict in the testimony of the undertaker and that of the husband. The undertaker swore that the husband had requested him to obtain strychnin, and he did so; while the husband swore that the request was for arsenic. The druggist did not keep any

record of poisons sold, and consequently could give no definite testimony on the subject. The husband testified that he and his brother suspended a teaspoonful of white arsenic in a teacupful of water, and injected one syringeful into the mouth and two syringefuls into the rectum. The syringe that he claims to have used was an ordinary Davidson bulb syringe with rectal tube attached.

One hundred and five days after death the body was taken up and the stomach and rectum placed in one jar, and a piece of the liver and one kidney in another. The jars were sent to Professor A. B. Prescott for analysis of their contents. It might be remarked here that the husband stated, when the officers came to remove the body, that he had embalmed it with arsenic. Dr. Prescott found in the stomach and rectum together about 20 grains of arsenious oxid, and from his analysis he calculated that the amount in the whole liver must have been from 6 to 15 grains, according to the size of that organ. Later, the body was again taken up, and the brain and a part of the muscles of the calf of one leg were sent to Professor Prescott for further analysis. In these Dr. Prescott failed to find any poison.

The question asked the experts on this trial, and the one on which the guilt or innocence of the accused seemed to hang, was in sum and substance as follows: Granted that white arsenic suspended in water was injected into the mouth and rectum a few hours after death, would it diffuse through the body to such an extent that it would be found in the liver and kidneys one hundred and five days after death?

One expert for the prosecution answered this question as follows: "If arsenic were injected into the stomach and rectum after death, it certainly would not reach the liver from the rectum, and I do not think it would reach the kidney. A small quantity of the arsenic might reach the portion of the liver in contact with the stomach. I do not think it would reach the kidney by diffusion after death. After death arsenic soon changes to an insoluble sulphid and ceases to diffuse. This would occur whether administered before or after death."

A second expert for the prosecution was very cautious in giving his opinion, as the following questions and answers will show:

"*Q.* If arsenic was put in the stomach after death, what, in your judgment, is the probability of its passing through into the liver by imbibition, or such portions of the liver as were not touched by the stomach?

"*A.* You must give me more factors than that to pass judgment on it. I must know whether the cavity of the abdomen was empty or whether it was full of fluid.

"*Q.* Suppose it was empty?

"*A.* I think if it was empty and the stomach was not lying upon the liver or touching the liver, there would be no imbibition to speak of.

"*Q.* Suppose it was not dry?

"*A.* It would pass. There would be imbibition from one organ to another, because chemists have demonstrated the fact that poisonous substances will pass through a bladder into the water on the opposite side. It is what we call dialysis."

Dr. Kedzie, for the defense, answered the question as follows: "I endeavored to find out whether arsenic would diffuse itself through dead matter in the same way that salt would. I tried to get the experiment in such a way that I could see the result. For this purpose I put a quantity of arsenic in a very dense solution of gelatin, which is an animal substance similar in properties to the tissues of the human body. This gelatin I put in the bottom of a large test-tube. I allowed this to cool so that it set into a solid mass. Then I took some more of the gelatin and put it into the tube, allowing it to cool and grow solid. In this last was put some hydrogen sulphid, so that if the arsenic should pass upward into the gelatin, it would be detected by the color. The two portions of gelatin did not mix; they simply came in contact. After a moment there was a little tinge of color where the substances came in contact, and the yellow line gradually widened. At first it was only as thick as a piece of paper, then in twenty-four hours a quarter of an inch, the next day higher, and so on."

It might be remarked parenthetically at this point that this experiment formed a beautiful and practical illustration of the diffusion of arsenic.

The writer, testifying for the defense, answered the question as follows: "If arsenic should be injected into the rectum and stomach after death, I should expect to find it in the adjacent viscera within twenty-four hours. I believe this from my reading and from direct observation. Taylor reports a case where a stomach was wrapped in a piece of wall-paper which had been colored with arsenic. The side of the paper next to the stomach was that which was not colored. The next day the contents of the stomach were analyzed, and found to contain large quantities of arsenic which had passed from the paper through the walls of the stomach. A week ago last Thursday I took from the body of a person who had been dead but a short time parts of the intestines. I tied one end of a portion of the intestine so that a fluid would not pass through it save by diffusion. I then injected the intestine with arsenic suspended in water, tied the upper end, and suspended the piece of intestine in a dilute solution of hydrogen sulphid, so that as soon as the arsenic should pass through the walls of the intestine it would show by its color. When I first put it into the fluid there was no coloration, showing that there was no arsenic on the outside of the intestine. After three hours the fluid on the outside was colored, showing that the arsenic had passed through the walls of the intestine into the surrounding fluid. I also took another portion of the intestine and placed arsenic in the same manner inside, then suspended this in a dry beaker, and in about the same time I detected arsenic on the outside of the intestine. If within twenty-four hours after the death of Mrs. Millard arsenic to the amount of one teaspoonful had been injected into the stomach and rectum, and the body buried and examined one hundred and five days afterward, from my reading, and the experiments just given, I certainly would expect to find the arsenic in the liver from that injected."

The evidence from the books on the point of postmortem diffusion introduced in the trial well illustrates the wisdom of excluding statements from books in the giving of testimony in criminal cases. At that time Taylor's work on poisons was regarded as the great authority upon the subject, and statements from this book were introduced as testimony in the case. In the earlier editions of this work the experiments of Orfila and Kidd, which fitted the case in hand, are referred to, but the conclusions drawn by Taylor are not justified by the facts he gives. He says: "The effects of cadaveric imbibition have been greatly exaggerated. Observation shows that it is too limited in extent to affect materially the conclusion usually drawn from the detection of poisons in the tissues. When a dead body is examined for a mineral poison, such as arsenic or antimony, shortly after death, and it is found in the liver or other soft organs, it is a fair inference that it was deposited in them during life."

It must be borne in mind that the use of arsenical embalming fluids was not so common in 1859, when Taylor wrote the above, as it was in 1882. But on another page he supposes that arsenic should be introduced into the stomach after death from natural causes, and concludes that even in such a case the expert would not be deceived, and could tell with certainty whether the poison was given during life or was introduced after death; a conclusion in which chemists of to-day cannot agree.

After the above-mentioned trial the writer concluded to investigate experimentally the question of the postmortem imbibition of arsenic. In this work he was aided by Dr. Dawson, then a student in the University of Michigan.

A large muskrat that had been caught by one of the students was killed and 3.24 grams of arsenious oxid suspended in cold water was injected with an ordinary bulb-syringe, with rectal tube attached, into the mouth and rectum. The rat was placed in a pine box and buried. After twenty-five days it was disinterred and the various organs removed and subjected to analysis, with the results shown in the following table:

Name of part examined.	Amount of arsenic, calculated as As_2O_3, found.
Kidneys	0.00095 gram.
Liver	0.01082 "
Lungs	0.19252 "
Stomach and contents	0.00686 "
Large intestine	0.40339 "
Small intestine	0.10157 "
Heart	0.02507 "
Brain	0.03960 "
Total As_2O_3 recovered,	0.78078 gram.

It will be noticed that the lungs contained a much larger amount of arsenic than the stomach. Evidently the larger portion of that injected into the mouth passed down the trachea instead of going down the esophagus—indeed, the amount found in the liver is larger than that found in the stomach. It is likely that the poison passed from the

lungs into the liver. The amount found in the brain is large, but in the muskrat the bones of the skull are thin in texture and not firmly united.

In a second experiment a cadaver was used. The person had been dead between two and three days, when the injections were made. A teaspoonful of white arsenic was suspended in cold water and was injected by means of a common bulb-syringe with rectal tube into the mouth and rectum. The body was placed in a dry cellar, and allowed to remain there for twenty-five days. The various parts as given in the table below were then removed, weighed, and subjected to analysis. In dissecting the body it was observed that, although the cuticle had decomposed to a certain extent, the internal organs were firm and remained in a fair state of preservation. This was true of all the parts removed except the brain, which was broken down to a semifluid condition.

The following table shows the part analyzed, its weight, the amount of arsenic estimated as arsenious oxid found, and the percentage of arsenic found in the various tissues :

Name of part taken.	Weight of part. Grams.	Weight of As_2O_3.	Percentage of As_2O_3.
Right kidney	104	distinct mirror	
Left kidney	90	0.00703	0.00782
Liver	865	0.08316	0.00961
Lower lobe of right lung	99	0.04333	0.04376
Heart	370	0.02199	0.00594
Transverse section of colon	85	0.02659	0.03128
Rectum	22	1.65000	7.50000
Spleen	48	0.00455	0.00947
Stomach	300	2.11200	0.70400
Brain	1028	0.00363	0.00035

It will be seen that while the right kidney contained only an unweighable quantity of arsenic, the left kidney furnished nearly as large a percentage as was found in the liver. I account for this by supposing that the liver caught up the greater portion of the arsenic passing down from the right lung, while on the left side the arsenic passed on into the kidney. Contrary to what was observed in the experiments on the muskrat, the stomach of the cadaver contained a large amount of arsenic, and it seems probable that some of the fluid thrown into the mouth passed directly into the stomach. I was somewhat surprised at finding arsenic in the brain, and the question arose, by what avenue did the poison reach this organ? It had been noticed that while throwing the fluid into the mouth at one time, when the bulb of the syringe was very forcibly compressed, a portion of the fluid returned through the nose. It is probable that some of the arsenic adhered to the roof of the pharynx and along the nasal passages, and from these penetrated the brain. However, arsenic placed in the stomach after death will diffuse in every direction and finally reach the brain.

Professor Kedzie, of the Michigan Agricultural College, at the same time, and wholly independently of the above work, made an experiment that he then reported to me as follows: "One of our students obtained

a cat that had been killed a few hours before by a gunshot wound in the head. Under my direction a quantity of arsenious oxid suspended in water was injected into the stomach and rectum, and the cat was then buried for thirty-one days. At the expiration of this time the animal was taken up, the liver, spleen, heart, and kidneys removed without contact with the contents of the alimentary canal, washed with water, and then oxidized with potassium chlorate and pure hydrochloric acid. The residue was reduced with pure zinc and sulphuric acid, and the metallic arsenic collected in a glass tube. From two-thirds of the liver 22 milligrams of metallic arsenic was obtained, equivalent to 0.53 of a grain of arsenious oxid for the entire liver. The heart, spleen, and kidneys were treated together, and from them I obtained 13 milligrams of metallic arsenic. There was thus obtained from this animal 35 milligrams of metallic arsenic, and if the whole of the liver had been used, there would have been 46 milligrams of metallic arsenic, equivalent to 0.89 of a grain of white arsenic obtained from viscera which could have received this arsenic only by postmortem diffusion from the contents of the alimentary canal. This result is directly opposed to the dictum of the older writers on medical jurisprudence, that imbibed arsenic in the viscera is proof of its administration before death."

Since one of the experts in the Millard case had given it as his opinion that arsenic would be converted in the alimentary canal into the sulphid, and that this would not diffuse, I made some experiments with this form of arsenic. The stomach of a dead cat was opened, and some moist sulphid of arsenic placed in this organ. The incision was carefully closed, and the animal buried. After sixty days it was taken up and arsenic was found abundantly present in the lungs, heart, liver, spleen, and kidney. The brain was not tested for the poison in this case.

The above-mentioned experiments have been confirmed by Miller, Witthaus, and others. In some of his experiments Miller provided against the direct absorption of arsenic from the pharynx into the brain. His statement is as follows: "The gullet was exposed by making an incision in the median line of the neck just below the larynx. Into the esophagus a longitudinal incision was made and a ligature placed immediately above the incision. Into the opening thus made a tube was passed through the gullet into the stomach. After the gullet was tightly secured to this tube the arsenical solution was poured down into this receptacle; the tube was then withdrawn, and the lower portion of the esophagus tied below the incision. Hence we are justified in claiming that all the poison recovered from any organ could not have reached that tissue in any other manner than by imbibition or a soaking process from the place of deposition—*i. e.*, the stomach. The spinal cord and brain were removed from every rabbit experimented upon before the thoracic or abdominal cavity was opened. The spinal cord was exposed in the usual manner by making two parallel incisions from the cervical to the lumbar region in the median line of the back, and the section

thus mapped out removed by sawing through the laminæ of the vertebræ. From a rabbit buried thirteen days the spinal cord, brain, contents of the urinary bladder, kidneys, and liver were removed. Owing to the special attention which we desired to give to the spinal cord, brain, and urine, these only were removed from a rabbit buried twenty-four days; also from a rabbit buried twenty-nine days the spinal cord and brain were taken. No urine was obtainable. These organs and secretions were placed in separate clean glass jars for chemical analysis." From the animals into which arsenic was thus introduced Miller obtained evidences of this substance in the brain, spinal cord, liver, kidneys, and urine.

He concludes his communication with the following: "On examining critically the preceding results we are doubtless justified in gleaning therefrom the following: That as the result of experimental research we have come in possession of indisputable evidence, demonstrated by strictly scientific methods, of the fact that when a poison is introduced into the stomach it can actually imbibe, soak, and diffuse itself into the various organs of the body, can be recovered from the liver, kidneys, spinal cord, brain, and interior of urinary bladder; also that the chemical evidence should not be held in the highest esteem and be given the place of first importance in all cases, as has hitherto generally been the case. It must be admitted that there are only very rare opportunities for the toxicologist to detect a discriminative method between ante- and postmortem poisoning. But the microscopist, with his knowledge of the histological and pathological appearance of organs, may perhaps be able to discover, by rigid searching with his microscope, some prominent appreciable difference; for it is not improbable that there may take place certain specific changes in the histological constituents of the organ due to the diffusion of a substance like arsenic, through the medium of the blood circulation during life, while these would not manifest themselves as the result of an after-death deposition."

Reese makes the following suggestions, which he thinks might be of benefit in determining whether a poison was administered before death or was introduced into the body after death:

"(1) A knowledge of the symptoms before death, where these are obtainable, will frequently throw much light on the case, although too much stress should not be placed upon symptoms merely, inasmuch as the symptoms of many diseases strongly resemble those of certain poisons. Thus, I have known several cases of fatal arsenical poisoning to have been mistaken for cholera morbus and treated as such by the attending physicians, and the certificate of death was made out, but in which I subsequently detected in the viscera large amounts of arsenic.

"(2) The chemical examination of the urine of the deceased. As is well known, the kidneys rapidly eliminate arsenic (and the same is true of other mineral poisons) from the body; and it is generally possible for the analyst to detect the poison in the urine both before and after death. Its discovery in this secretion may, I think, be regarded as very conclusive evidence of antemortem poisoning; for although it

ferric chlorid. He found that after a few days the ferrocyanid had penetrated every part of the body. He also experimented upon warm-blooded animals. Some of his results may be condensed as follows: He introduced one-half gram of arsenite of potassium dissolved in 200 grams of water into the stomach of a dead dog. The body was allowed to lie for seven days on its left side. At the expiration of this time chemical analysis showed the presence of arsenic in the brain, liver, lungs, and heart. In a second experiment he introduced 150 c.c. of a solution of arsenite of potassium into the stomach of a dead rabbit. This animal was placed on its right side. A chemical analysis made eighteen days later showed traces of arsenic in the brain, a large quantity in the liver, lungs, and heart. In a third instance he placed a considerable quantity of the same solution in the large intestine of a rabbit. An examination eight days later showed no arsenic in the brain and cord, doubtful traces in the lungs, heart, and kidneys, but a very marked quantity in the liver. In still another experiment, after the introduction of the arsenic, the animal was suspended by its hind legs. After seventeen days arsenic in large amount was found in the liver and a smaller amount in the brain. In still another experiment the animal was suspended by its forelegs after the introduction of the arsenic. After eighteen days no arsenic was found in the brain and a small quantity in the liver. A large quantity of the arsenite of potassium was placed in the stomach of a dead dog. Forty-eight hours later a marked trace of arsenic was found in the brain, and large quantities in the liver, kidney, heart, and lungs. In one experiment the animal was not examined until one hundred and ninety-seven days after the introduction of the poison. The cranial cavity contained decomposed brain tissue in which arsenic was found in abundance.

Torsellini concludes from his experiments that arsenic introduced into the stomach after death may be found in the brain within from six to seven days, in the liver somewhat earlier, and in the lungs and heart earlier still. He also concludes that the position of the body after death determines the direction and rate of diffusion. When the analysis is not made until many months have passed, he decides that it is impossible to distinguish by chemical analysis between antemortem administration and postmortem diffusion.

In a case of trial for murder by the administration of arsenic Schaitter gave his evidence in favor of the belief of antemortem administration, notwithstanding the fact that only traces of arsenic were found in the liver, because the body that was exhumed five and one-half months after death was found to be distinctly mummified, and because vomiting and purging were symptoms observed during the last illness. While a discussion of the mummifying properties of arsenic does not come within the scope of this paper, it may be stated that mummification in and of itself is no positive proof of arsenical poisoning. Many mummified bodies contain no arsenic, while, on the other hand, many arsenical bodies rapidly decompose. Moreover, arsenic introduced into

and both kidneys free from arsenic, while the liver contained a large amount. The conclusions which they draw from their experiments may be stated as follows: (1) Different substances, such as arsenic, diffuse through the dead body. This diffusion goes on slowly and extends from one organ to that immediately adjacent, and from this on to the next. (2) In consequence of this diffusion such substances as arsenic may after some days diffuse from the stomach into the adjacent organs. (3) If the left kidney contains arsenic while the right contains none, it may be inferred that the poison is diffused from the stomach after death. If both kidneys contain arsenic during the first week after death, its administration during life may be assumed. (4) A separate examination of the left and right lung, also of the left and right portions of the liver, may aid in determining whether the poison has been introduced during life or after death. (5) Arsenic does not diffuse from the stomach into the brain within four weeks. From the pharynx it may pass into the brain in a shorter time. (6) If the poison has been introduced into a part of the dead body other than the stomach, an examination of the place of introduction and of tissues more or less distant should be made.

While most of the experiments on the postmortem imbibition of poisons have been made with arsenic, it must not be inferred that this substance diffuses through the body more readily than other poisons. All crystalline substances will diffuse through animal tissue.

It will be seen from the evidence here presented that it is highly desirable that some steps should be taken to prevent the indiscriminate and common introduction of poisons into the body after death. No undertaker or any one else should be allowed to embalm a body until authorized to do so by the attending physician, and when a body is embalmed, a record of the chemical composition and amount of the embalming fluid used and the method of its administration should be kept. Attempts have been made in various States in the Union to have the embalming of bodies regulated by law. Up to the present time, however, so far as I know, nothing has been done in this direction.

[1] In Michigan the license of an embalmer may be annulled if he employs an arsenical embalming fluid.

Each article received by the expert should also be carefully described, together with the detailed statement of the results of the preliminary examination in each case.

The most important kinds of stains that are submitted for medicolegal examination are:

1. Blood-stains.
2. Stains containing blood, such as menstrual, lochial, and nasal, after epistaxis.
3. Seminal stains, as in cases of alleged rape, adultery, or sodomy.
4. Stains containing mucus or pus, such as nasal, leukorrheal, and gonorrheal.

BLOOD-STAINS.

The expert is more frequently required to examine stains medicolegally for blood than for anything else, and he is requested to determine, in the first place, whether the stain is a blood-stain or contains blood or not. Second, if a blood-stain, whether the blood came from a mammal or from a bird, fish, or reptile. Third, if the blood were mammalian, is it consistent with that of any given species of mammal. It is often alleged by the accused in cases of homicide that the blood-stains in question emanated from some animal, or that they were made by the blood of a fish, bird, or reptile. In such cases the work of the expert is narrowed down to determining whether the blood in the stain is consistent with that of the animal mentioned, or consistent with its being human blood. When no data are given, the expert must give the result of his investigation, and state whether or not these results are consistent with the staining being that of human blood.

There are also some other questions that the expert may be required to decide in cases in which there exists no doubt that the blood in the stain has emanated from a human being, as, for instance, in the case of blood-stains or pools of blood found in the immediate neighborhood of the victim of a homicide. It may be possible to determine approximately the length of time that has elapsed between the escape of the blood from the blood-vessels and the finding of the body of the victim from the observation of the stains in question. Again, an opinion is often requested concerning the direction from which the blood must have come in order to have produced a given stain. In order to throw light upon these questions it is necessary to understand some of the physical and chemical properties of the blood.

The blood is composed of a very pale, straw-colored fluid having a specific gravity of about 1028. In this fluid are suspended a very large number of small solid bodies—the red and the white blood-cells or corpuscles; also a number of smaller solid bodies called the blood-plates, which, up to the present time, have no medicolegal importance. The blood plasma is made up of the blood-serum and the fibrin, the latter resulting from the union of two albuminous substances called the fibrinogeneous substance and the paraglobulin. This union constitutes the so-called coagulation, which occurs after the escape of the blood from the

late in one and one-half minutes; that of the rabbit, sheep, and pig in from one-half to one and one-half minutes; that of the horse and ox in from five to thirteen minutes; that of the dog in from one to three minutes; that of man in from three to four minutes. The coagulation is complete in human blood in from nine to ten minutes. In the case of bird blood, coagulation is rapid and the coagulum large, while in cold-blooded animals the coagulation is slow and the coagulum small.

The following circumstances increase the rapidity of the coagulation: A slight increase of the temperature; exposure of a large surface of blood to the air—as, for instance, when it is collected in a shallow dish; contact with numerous points of some foreign body, as when

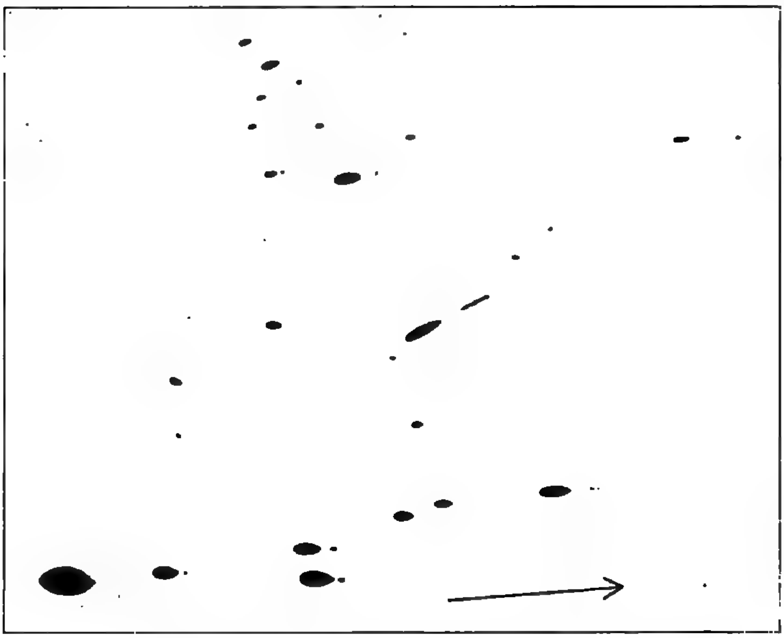


FIG. 78.—Showing direction from which the blood came.

stirred or beaten with a number of wires, or, as in the case of many stains submitted to medicolegal examination, when a small drop falls upon a rough surface. The following conditions may delay, or even prevent, the coagulation: Exposure to cold, as, for instance, if cooled rapidly to the freezing-point, coagulation will not take place for an hour or so; contact with oily or greasy substances; admixture with syrupy liquids; and heating the blood quickly to a temperature of 56° C. (132.8° F.) will entirely prevent coagulation.

On account of its coagulability the blood has the property of a viscid fluid, and this viscidity often renders it possible to determine in certain blood-stains the direction from which the blood must have come. When a minute drop of blood strikes an object after having been pro-

pelled from an artery, it forms an oval or pear-shaped stain if the surface against which it impinges be more or less oblique, and the bulk of the blood is driven to the farther end of the stain, which in the case of the pear-shaped stain forms the stem of the pear; the same results obtain by the force of a blow caused by some blunt instrument, such as a club, striking in a pool of blood. If the surface be horizontal, whatever may be its nature, and remain so until the stain has become dry, it can readily be seen by inspection that the pointed or stem portion of the pear-shaped stain contains the largest quantity of the blood. If, however, the surface against which the blood impinges be vertical and of such a nature that it does not immediately absorb the blood, then the bulk of the blood, which has been driven to the farther part of the stain, will, if the blood came from below upward, gravitate back to the lower portion. If the surface happen to be a rough one, like that of a shaggy overcoat cloth, the coagulation takes place so rapidly that this gravitation does not occur, so that, if a drop of blood be propelled from near the surface of the ground upward against the surface of a rough overcoat, that surface being vertical, it will be found on examination after the stain has become dry that the bulk of the blood is in the upper portion of the stain, which is generally the smaller end, like the point or the stem in a pear-shaped stain. The importance of being able to determine the direction from which the blood came in order to form the stains found in certain cases is self-evident and needs no further discussion. The accompanying illustration (Fig. 78), reproduced from a photograph, shows the above-mentioned points.

Drying of Blood.—The time required for blood to dry is also important in certain cases. For instance, in a case of double murder recently tried in Massachusetts the appearance ot the blood beneath the two bodies showed that a considerable length of time must have elapsed between the deaths of the two individuals. A drop of blood dries much more slowly than a drop of water of the same size. The character of the surface upon which the blood falls also has considerable bearing upon the time required for the complete drying of the blood. If the surface be an absorbent one, so that the blood may permeate the substance itself, as in the case of a piece of unstarched cotton or linen cloth, the drying takes place quite rapidly, but if the surface be a non-absorbent one, like that of a piece of glass or a board having a smooth surface, and also in the case of some kinds of dense woven fabrics, the time required for the drying of a drop of blood is much greater than that of a drop of water of equal size. A large drop of blood requires proportionally a longer time to dry than a small drop. For instance, a drop of blood allowed to fall from the end of a finger on to a piece of glass, the drop measuring $\frac{3}{8}$ of an inch in diameter, required one hour and five minutes for complete drying under favorable conditions—namely, in a room with an open fire and a temperature of 71° F. A drop of similar size on a piece of soft pine board in a room in which the temperature was only 65° F., and with the atmosphere not so dry as in the previous instance, did not undergo any perceptible change for half an

hour, and in one hour it had only begun to shrink and have a glazed appearance on the edge. This glazed appearance gradually extended toward the center of the drop, and the drying became complete in exactly two hours. Therefore, from one to two hours is required for a single drop of blood to become dry.

The above facts show how necessary it is for the expert carefully to observe every blood-stain in the medicolegal cases that are submitted to him for investigation. He must observe and make note of their number, exact location, shape, color, and whether or not the individual stains have a different density in different parts. From the facts obtained by simple inspection, which should be made as quickly as possible after the receipt of the substances to be examined, important information may possibly be gained with reference to the age of the stain, the direction from which the blood emanated, and perhaps some idea of the force with which it was propelled.

Color of Blood-stains.—If a blood-stain be recent, it can sometimes be determined, within narrow limits, by carefully observing its color, how long a time has elapsed since the stain was made. A freshly made blood-stain, after it has become dry, under ordinary circumstances has a bright scarlet color. Upon exposure to ordinary daylight this color gradually changes to a dull brown during a period of about ten days, after which time the color does not change materially; therefore if a given stain has been exposed only to light and air under ordinary conditions for less than ten days, some opinion can be formed as to the approximate age of the stain. It must be borne in mind, however, that there are some conditions that hasten this change of color: continuous exposure to direct sunlight; the application of a high degree of heat, such as might be obtained by the application of a hot iron or the heating of the substance containing the stain on a stove or in an oven; or treating the stain with certain preservatives, like alcohol or naphtha, will very rapidly produce the change of color to a dull brown.

METHODS OF IDENTIFYING BLOOD-STAINS.

The methods employed in the identification of blood-stains are: (1) Chemical; (2) optical; (3) microscopic examination; (4) the biologic or serum test.

The chemical and optical methods are employed for the purpose of detecting the red coloring-matter of the blood—hemoglobin—or its decomposition products in those cases in which it has been decomposed by conditions to which the stain has been subjected. They serve also to detect the serum-albumin contained in the stain, although the detection of this constituent serves merely as a confirmatory test and is of comparatively little value, since albumin is a constituent of many other fluids. Moreover, our tests, under the conditions that obtain in testing stains medicolegally, do not enable us to distinguish between the serum-albumin of the blood and egg-albumen.

The detection of the hemoglobin, however, shows us with certainty

the presence of blood, but since hemoglobin is contained in the blood of many animals, its detection throws no light upon the nature of the animal from which the blood in any given stain emanated. To determine this latter question resort is had to the microscopic examination of the blood-cells in the stains and to the serum-test. By means of these methods of investigation it is often possible to determine whether the blood originated from a human being or from certain animals.

Before considering these methods of investigation in detail it will be necessary to consider briefly the more important properties of hemoglobin and its decomposition products.

Hemoglobin and its Decomposition Products.—Hemoglobin is the red coloring-matter of the blood, and is found in the blood of all the vertebrates with the exception of two, the *amphioxus* and *leptocephalus*, and in that of many of the invertebrates. It is the substance that carries the oxygen to the tissues, and with the oxygen it forms two compounds, oxyhemoglobin and methemoglobin. In the oxyhemoglobin the oxygen is very loosely combined with the pigment and separates from it very easily, while in methemoglobin the combination is much more stable. Solutions of oxyhemoglobin have the bright cherry-red color, such as is seen in arterial blood, while the darker color of venous blood is that of a solution of hemoglobin deprived of its oxygen. The color of a solution of methemoglobin is brown. Solutions of these three substances when examined with the spectroscope show different absorption-bands, which will be spoken of later in relation to spectroscopic tests for blood.

Hemoglobin contains iron; it also forms distinct compounds with nitric oxid (N_2O_2) and carbon monoxid, the solutions of which give characteristic absorption spectra. These compounds, however, have no medicolegal importance in the examination of blood-stains. The carbon monoxid hemoglobin has medicolegal importance, however, in cases of poisoning by illuminating-gas and by the fumes from burning charcoal; poisoning by illuminating-gas is quite commonly seen at the present time, owing to the large percentage of carbon monoxid in the illuminating-gas used in our large cities. It is this compound that imparts the bright cherry-red color to the blood and tissues in cases of poisoning by this gas.

Hemoglobin is quite easily decomposed by various agencies. If a solution of it be heated to boiling, the color changes to a brown, due to the decomposition of the hemoglobin into a brown pigment—hematin—and a proteid—globin. This pigment hematin is quite important medicolegally because it may be formed from hemoglobin by many agencies to which blood-stains may be exposed, and also because it is formed during the performance of some of the principal chemical tests for blood.

Hematin contains all the iron of the hemoglobin from which it was derived. It is insoluble in water, alcohol, and ether, but is soluble in alkaline hydroxids and in alcohol containing sulphuric acid. Solutions of hematin give characteristic absorption spectra that will be described later. If a solution of hematin be treated with ammonium sulphid or

some other reducing agent, a pigment called hemochromogen is formed; this pigment was formerly designated reduced hematin.

Another decomposition product that is of importance in the medico-legal examination of some blood-stains is hematoporphyrin, or iron-free hematin. This pigment is produced from hemoglobin or hematin by the action of those agencies that are capable of removing the iron from these compounds; some of these agencies are conditions to which blood-stains may be subjected accidentally or intentionally, as has been shown by Liman, Kratter, and Hammerl.[1] When this decomposition has taken place in a blood-stain, the application of the ordinary tests will not reveal the presence of blood pigment, and it becomes necessary to resort to special methods for the recognition of the hemochromogen or the hematoporphyrin by means of the spectroscope.

The most important agent causing this decomposition is heat. If a blood-stain be heated to a temperature of 142° C. (287.6° F.) for a period of one hour, or to a higher degree for a shorter period of time, the pigment will be so decomposed that it cannot be detected by the hemin or the guaiacum tests. Practically this may happen to a blood-stain if the fabric containing it be ironed with a hot iron. If the stain be dry, the application of a high degree of heat will not destroy the red blood-cells, but, on the contrary, will fix them, so that they will not afterward be destroyed by water or other agents that ordinarily quickly destroy the red blood-cells. If, however, the stain be wet when the heat is applied, the red blood-cells will be destroyed even at a temperature of 56° C. (132.8° F.). When this decomposition has taken place, the pigment can be dissolved out by concentrated hydrochloric, sulphuric, or glacial acetic acids; an alcoholic solution of sulphuric acid is capable of dissolving a sufficient amount of this pigment to permit of its recognition by the spectroscopic examination.

The writer has also found that if blood-stained articles be treated with certain germicides, such as naphtha or benzene, or certain deodorizers or disinfectants, such as aluminum chlorid or the so-called bromo-chloralum, the blood-pigment will be so decomposed that it cannot be detected by the ordinary chemical tests. At the same time the red blood-cells will be fixed to such an extent that they will resist subsequent treatment with water.

Chemical Tests for Blood.—The principal chemical tests for blood are the following:

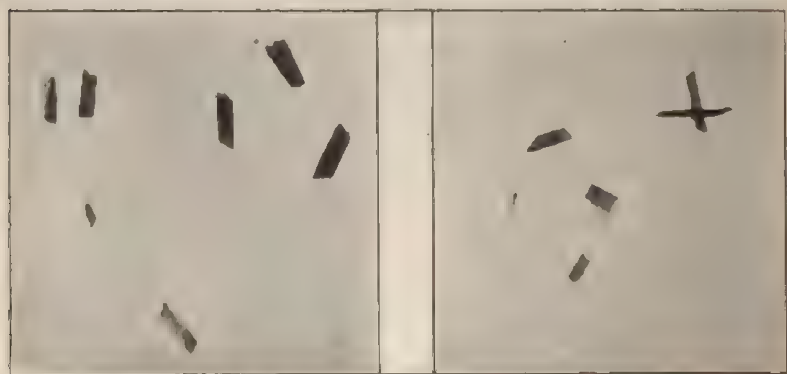
1. Teichmann's, or the hemin, test.
2. Sodium tungstate test.
3. Guaiacum test.
4. Albumin test.

Teichmann's Test.—This is by far the most important test for blood-pigment, and is extremely delicate. It may be performed in the following manner:

If the object to be examined be a dried blood-stain, a small fragment of the dried blood should be removed from the stain with the

[1] *Vierteljahresschr. f. ger. Med.*, 1892, vol. iv., pp. 41–61, 62–75.

point of a knife and transferred to a glass slide. If the stain be a diffused one, or if the blood, while still fresh, had soaked into the fabric, as in the case of a stain upon cotton or linen cloth, so that a fragment of the dried blood cannot be removed by the knife as above, then it suffices to scrape a small portion of the stain with the knife-point, collecting the dust thus removed upon a glass slide. The fragment of dried blood or the dust obtained in the above-mentioned way should then be treated on the slide with a small drop of water in which has been dissolved a minute fragment of sodium chlorid (common salt) or some other halogen salt, such as potassium iodid or bromid. This drop should then be evaporated to dryness by very gentle heat, the dried residue covered with a cover-glass, a drop of glacial acetic acid allowed to run under the cover-glass, and the slide again gently heated until bubbles of gas are seen to form in the liquid under the cover-glass. This shows that the glacial acetic acid has been heated to the boiling-point. If, now, the slide be allowed to cool, an examination with the microscope



FIGS. 79, 80.—Hemin crystals (common forms) (× 750).

will reveal the characteristic crystals of the chlorid of hematin in case the stain examined contained blood. These crystals of the chlorid—or iodid in case potassium iodid has been used—of hematin are called "hemin" crystals, and they have a very characteristic crystalline form.

The normal hemin crystals have a yellow to chocolate-brown color, and separate in the form of small rhombic plates. These crystals naturally vary a little in size according to the rapidity of their formation. Sometimes two or more arrange themselves in the form of a cross or a rosette. Sometimes, particularly if the fragment of dried blood upon the slide was of considerable size, the form of the crystals in some parts of the preparation may be somewhat modified, some of the crystals assuming a pointed, oval shape, and in some the outlines may be a little irregular; in all cases, however, a sufficient number of the normal perfect crystals will be seen to render their identification positive. The accompanying illustrations, reproduced from photographs taken from different portions of the same slide, show this variation (Figs. 79 and 80).

This test depends upon the principles, first, that hematin is formed from the decomposition of the hemoglobin by the heat, and, secondly, that the hematin in solution in the boiling glacial acetic acid unites with the chlorin of the salt to form the chlorid of hematin, which is soluble in boiling glacial acetic acid, crystallizing from this solvent on cooling.

Precautions.—Care should be taken in heating the slide in the performance of this test not to raise the temperature so high as to decompose the hematin in the first dry residue obtained. If the temperature be raised to about 142° C. (287.6° F.), no hemin crystals will be formed, as has been shown by Hammerl (*loc. cit.*).

Further, on heating the slide after the addition of the glacial acetic acid, the temperature should not be raised so high as to produce active boiling of the acid, since active ebulition may carry all the pigment beyond the edge of the cover-glass, which might prevent the detection of the hemin crystals.

The hemin test will not serve to detect blood-pigment in those blood-stains that have been heated to a high temperature, that have been subjected to the prolonged action of naphtha, a solution of aluminum chlorid, or bromochloralum, or that have been exposed for a long time to direct sunlight.

The Sodium Tungstate Test.—This test is of great value in the case of diffused blood-stains caused by the action of water upon the original stain, as in unsuccessful attempts to wash a blood-stain from cloth, and in cases in which the blood exists in solution in water or some other aqueous fluid, such as urine. By means of this test all the blood-pigment is precipitated from its aqueous solution, and thus concentrated to a small volume in the form of a precipitate that can afterward be tested by the hemin test or subjected to spectroscopic examination.

In the case of a diffused blood-stain in which the blood-pigment has been largely diluted with water and spread over a large surface of cloth or other fabric, the application of the hemin test in the ordinary way will generally fail to detect the blood-pigment. It is, therefore, necessary first to remove all the blood from a considerable piece of the cloth, concentrate to a small volume, and identify it by one or more of the other tests. This is best done in the following manner: A piece of the cloth of sufficient size—the amount required depending upon the extent to which the blood has been diluted, this being determined generally by simple inspection—is removed and placed in an evaporating dish or wineglass containing distilled water in which has been dissolved a crystal or two of potassium iodid. In this the fabric is soaked out and frequently stirred and pressed by means of a glass rod, so as to facilitate the thorough washing-out of the pigment from the cloth. The fluid should be poured off into a flask or test-tube, according to the amount used, and the process repeated once or twice with fresh portions of water and potassium iodid, and finally the fluid entirely pressed out from the cloth. These united washings should then be filtered so as to remove all solid particles, strongly acidulated with acetic acid, and

of these instruments the reader is referred to the works on optical apparatus or to the catalogue of the manufacturers of microscopes and microscopic accessories.

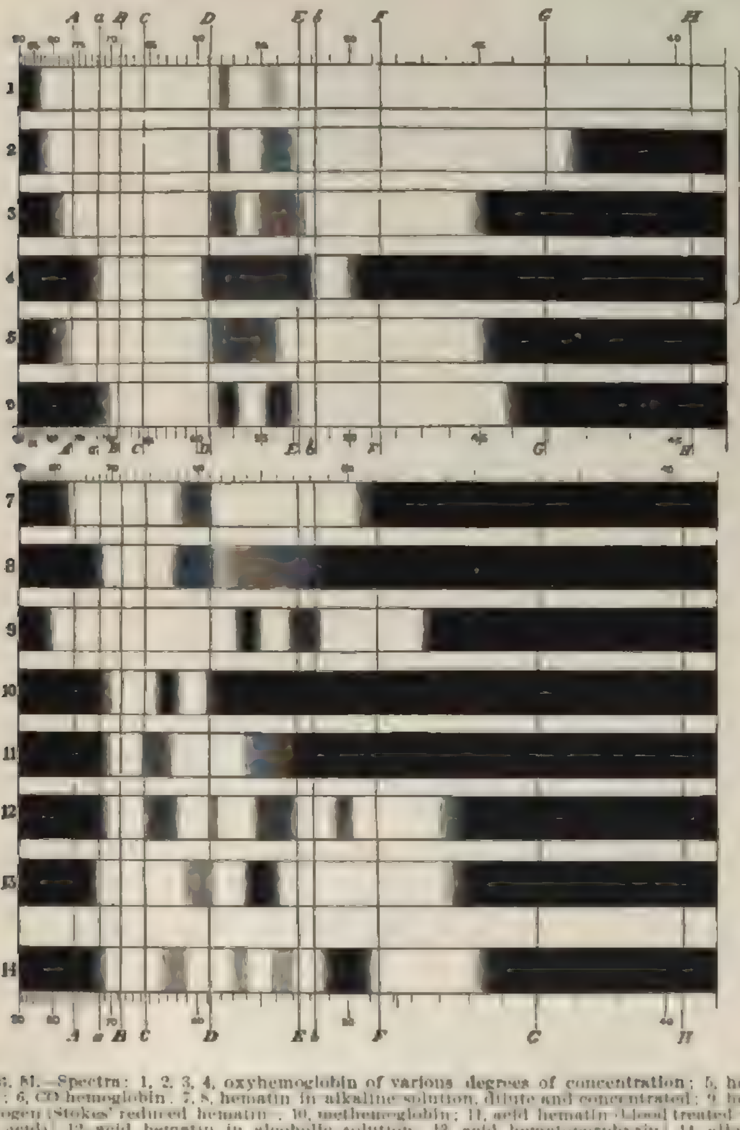
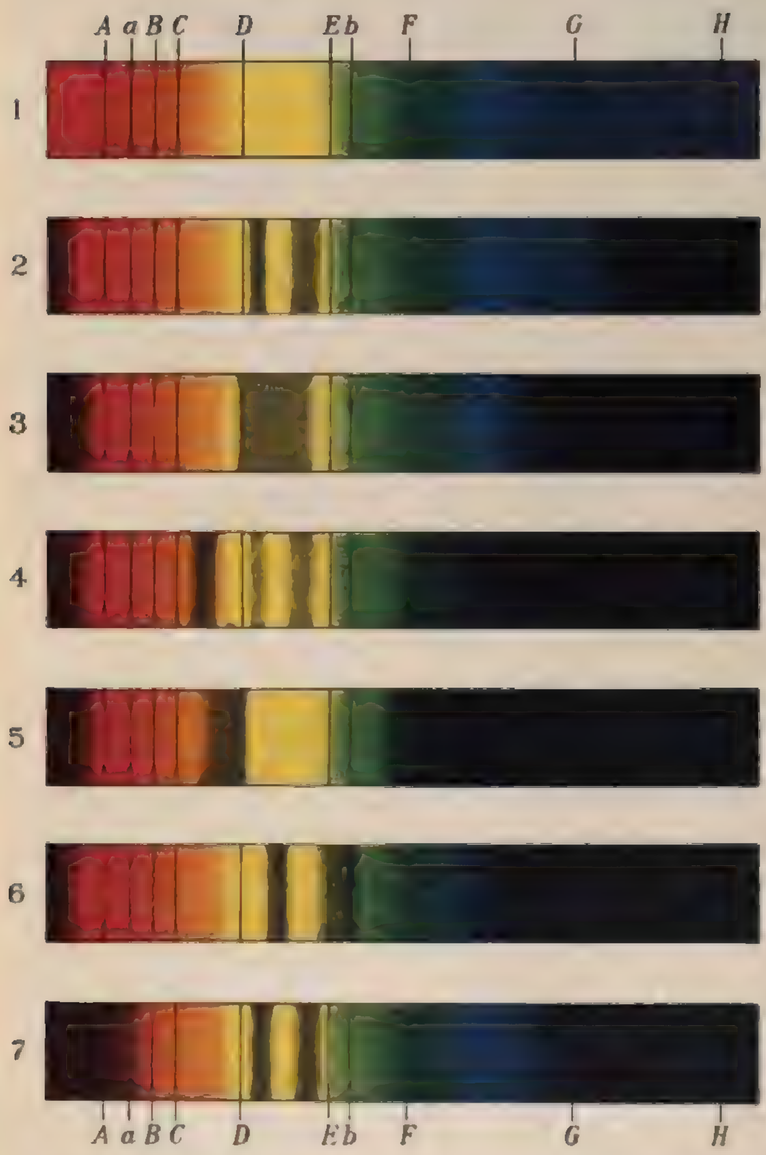


FIG. 81.—Spectra: 1, 2, 3, 4, oxyhemoglobin of various degrees of concentration; 5, hemoglobin; 6, CO hemoglobin; 7, 8, hematin in alkaline solution, dilute and concentrated; 9, hemochromogen (Stokes' reduced hematin); 10, methemoglobin; 11, acid hematin (blood treated with acetic acid); 12, acid hematin in alcoholic solution; 13, acid hematoporphyrin; 14, alkaline hematoporphyrin.

Oxyhemoglobin.—The characteristic spectrum of oxyhemoglobin can be seen only in very dilute solution. If a solution containing about 0.75 per cent. be examined in a layer 1 cm. (0.4 inch) thick, most of the rays of light will be absorbed between the lines *D* and *b*, thus forming

PLATE 12.

BLOOD SPECTRA.



1. Solar spectrum.
2. Oxyhemoglobin.
3. Hemoglobin.
4. Methemoglobin in dilute solution.
5. Hematin in alkaline solution.
6. Hemochromogen, or reduced hematin.
7. Carbon-monoxid hemoglobin.

of oxyhemoglobin, except that the two bands lie a little nearer the violet end of the spectrum. It can be distinguished from the oxyhemoglobin spectrum, however, by the fact that it remains unchanged on heating the specimen under examination with ammonium sulphid (see spectrum 6, and Plate 12, No. 7).

Hematin.—The recognition of this pigment is of importance in those cases in which the stain has been subjected to the action of those agencies that cause decomposition of the hemoglobin (see above). In order to extract the hematin from a blood-stain so as to prepare it for spectroscopic examination it is necessary to treat the stain, or a portion of it, with a dilute alkali (sodium or ammonium hydroxid), with glacial acetic acid, or with alcoholic solution of sulphuric acid, since hematin is insoluble in water, alcohol, and ether.

The acid and the alkaline solutions of hematin give different spectra. The spectrum of hematin in alkaline solution has a dark band between *C* and *D*, lying near *D*, and if sufficiently concentrated, overlapping *D*, while the violet end of the spectrum will be almost absorbed (see spectra 7 and 8, and Plate 12, No. 5). The spectrum of hematin in acid solution has an absorption-band between *C* and *D*, very close to the line *C* (see spectrum 11). In an alcoholic sulphuric acid solution properly diluted the spectrum may have four bands, one between *C* and *D*, nearer *C*; a very faint band, not always visible, just to the right of *D*; a broad band between *D* and *E*, nearer *E*; and the fourth band between *b* and *F*, a little nearer *b* (spectrum 12).

Hemochromogen or Reduced Hematin.—This is produced by treating an alkaline solution of hematin with a reducing agent, such as ammonium sulphid or Stokes's reagent. Its solutions give a well-defined absorption-band midway between *D* and *E*, and a fainter band between *b* and *E* (see spectrum 9, and Plate 12, No. 6). On exposure to the air an alkaline solution of hemochromogen absorbs oxygen and is reconverted into hematin.

Hematoporphyrin.—This is formed by dissolving blood-pigment in concentrated sulphuric acid or by heating it with concentrated hydrochloric acid. The solution in concentrated sulphuric acid gives a spectrum having two bands, one faint and to the left of *D*, and the other more distinct, between *D* and *E* (see spectrum 13). In dilute alkaline solution its spectrum has four bands, one faint and midway between *C* and *D*, the second and third rather faint, and having a position similar to the α and β oxyhemoglobin bands; and the fourth, a broad dark band reaching from *b* nearly to *F* (spectrum 14).

Method of Examination.—The method of treating a blood-stain for spectroscopic examination varies according to the quantity of material at disposal and according to the age of the blood-stain—that is, whether the hemoglobin is present in the stain unchanged or has been decomposed by agencies mentioned above. If plenty of material be available and the blood-pigment be mostly undecomposed, a portion of the stain can be treated with a little water or dilute salt solution, and examined directly with the direct-vision spectroscope, and the oxyhemo-

ticles of the reddish substance, causing them to fall near the middle of a large, thin glass cover. Apply in close proximity to them a very small drop of three-fourths per cent. salt solution, bring the particles of supposed blood-clot to its edge, and proceed as I have already directed.

"After thus examining the spectrum of the substance you may generally, by rotating the stage, cause the colored fluid to partly drain away from the solid portion, wherein, under favorable circumstances, should the specimen be blood, the granular white blood globules become plainly visible, as do also cell walls of the red discs. Among the latter, if your mental and physical vision be keen enough, you can, by the aid of a $\frac{1}{25}$ immersion lens and an eye-piece micrometer, measure a series of corpuscles accurately enough to discriminate human blood from that of an ox, pig, horse, or sheep."

Since there are a few substances solutions of which give a spectrum somewhat resembling that of oxyhemoglobin, such as solutions of alkanet root in alum and those of cochineal, the spectroscopic examination should not be restricted to the recognition of the spectrum of oxyhemoglobin alone. The stain should be treated with the different reagents, so as to obtain several of the blood spectra. The solution of oxyhemoglobin should, after the oxyhemoglobin bands have been recognized, always be converted into hemoglobin and again examined, since the vegetable solutions mentioned above are not changed by the action of reducing agents.

The methods of examination heretofore described enable us to determine simply the presence of the coloring-matter of blood, which throws no light whatever upon the kind of blood, since the red coloring-matter —hemoglobin—is contained in the blood of all vertebrates except the *amphioxus* and *leptocephalus*,[1] and in that of many of the invertebrates.

In order to obtain any information as to the nature of the animal from which the blood in question originated, resort must be had to the microscopic investigation, for the purpose of determining, if possible, the form and size of the red blood-cells, and to the serum test.

Microscopic Examination of Blood.—The red blood-cells differ in different animals. In birds, reptiles, and fishes it is oval in shape and has a distinct nucleus, while in most mammals it is a circular, biconcave disc without a nucleus; in the camel and llama tribe, however, the red cells are oval in shape, but have no nucleus. The distinction between any kind of blood having nucleated red cells and one having non-nucleated red cells offers no difficulty whatever, even in the case of dried stains, since the nuclei are very stable and not readily destroyed, and can be very easily recognized by their high density. The microscopic distinction between the different kinds of mammalian blood presents much greater difficulty, since, with the exception of the camel tribe, the only difference is in the size of the red blood-cells. This difference in size lies within such very narrow limits that in some cases the positive distinction, by the microscope, between the blood of certain

[1] Lankester, *Proc. Royal Soc.*, 1872, vol. xxi., p. 71.

object micrometer consisting of a glass slide upon which are ruled lines exactly $\frac{1}{100}$ of an inch apart, one $\frac{1}{100}$ inch space being divided into ten subdivisions by ruled lines; each subdivision, therefore, is exactly $\frac{1}{1000}$ of an inch. It is necessary to subject each scale to exact scientific examination to determine its accuracy by comparison with other micrometers known to be accurate. Having determined the accuracy of the object micrometer, it is very easy to determine the valuation of the spaces on the eye-piece micrometer. The object micrometer being placed upon the stage and the scale accurately focused, the line upon one side of the standard $\frac{1}{1000}$ inch space is brought into exact juxtaposition with one of the long lines on the eye-piece scale; then the number of divisions on the eye-piece scale exactly covering the $\frac{1}{1000}$ inch space on the object micrometer are counted. If, for instance, it take just eighteen divisions of the eye-piece scale to cover exactly the $\frac{1}{1000}$ inch space on the object glass, it means obviously that each division of the eye-piece scale corresponds to $\frac{1}{18000}$ of an inch. If, therefore, in measuring any object in focus upon the object glass,—as a blood globule, for instance,—it be found that six divisions of the eye-piece are required to cover exactly the object, it becomes evident that that object measures exactly $\frac{6}{18000} = \frac{1}{3000}$ inch.

The cobweb eye-piece micrometer, instead of containing a ruled glass slip, consists of two very fine threads or cobwebs drawn across the field and fastened in such a way that one of the cobwebs is fixed while the other is movable by a screw. The thread of the screw is generally so cut that a complete turn of the head will move the thread $\frac{1}{100}$ inch. The head of the screw is accurately graduated into 100 divisions, and a scale upon the side of the eye-piece indicates the number of complete revolutions of the head of the screw. The valuation of the space between the two cobwebs for each complete revolution of the wheel, or for each fraction of a revolution, can be determined in the same way as the valuations of the eye-piece divisions in the Jackson micrometer. By bringing the fixed thread in exact juxtaposition with one line of the $\frac{1}{1000}$ inch space on the object micrometer slide and then turning the screw until the movable thread exactly covers the line at the other side of the $\frac{1}{1000}$ inch space, the reading of the scales will show the distance in revolutions of the screw required to move the movable thread over the $\frac{1}{1000}$ inch space. From this the exact valuation of a complete revolution of the screw, or any fraction thereof, can easily be calculated.

As to the structure of the red blood-cells, authorities differ. Many of the older physiologists, as Hewson,[1] Wells,[2] and Schwann,[3] and more recently the late Dr. Richardson,[4] considered that the blood-cells had a distinct cell-wall containing a fluid colored with the red pigment, while others, among whom were Brücke,[5] Rollett,[6] Kollmann, and later For-

[1] *Hewson's Works*, Sydenham Soc., London, 1846.

[2] "On the Color of the Blood," *Philada. Trans.*, 1797, p. 429.

[3] *Microscopic Researches*, Sydenham Soc., 1847, p. 67.

[4] "On the Value of High Powers in the Diagnosis of Blood-stains," *Amer. Jour. Med. Sci.*, July, 1874.

[5] "Die elementar Organismen," *Sitzungsber. d. k. Akad. Wien*, vol. xliv., pt. xi., p. 387.

[6] "On Blood," *Stricker's Handbook*.

mad,[1] thought that they consisted of a stroma or network that is colorless, elastic, and made up of an albuminous substance the interstices of which contain the coloring-matter. This latter theory appears to be supported by the weight of evidence.

When the red blood-cells of mammalian blood are treated with water or with any fluid the density of which is less than that of the blood-serum, they undergo a distinct change in form, owing to the absorption of water and the loss of their own contents by osmosis. They first lose their biconcavity, then become biconvex, and finally spheric. While this change is taking place the distance between the two edges diminishes, so that finally the diameter of the cell, which has become spheric, is only about two-thirds of the original diameter when the cell had its normal biconcave shape. At the same time its density has disappeared, so that it is much more difficult to see, the outline of the cell being a mere line. Finally, if this influence continues, the cells become entirely destroyed. This diminution in the diameter of the swollen blood-cells is quite constant in all mammalian blood, as shown by the following measurements made by Dr. Formad:

	Average diameter of spheric corpuscles.	Average diameter of normal corpuscles.	Normal diameter reduced one third.
Man	1-4300 inch.	1-3200 inch.	1-4267 inch.
Guinea-pig	1-4500 "	1-3400 "	1-4533 "
Wolf	1-4600 "	1-3450 "	1-4580 "
Dog	1-4800 "	1-3580 "	1-4773 "
Rabbit	1-4900 "	1-3662 "	1-4882 "
Ox	1-5600 "	1-4200 "	1-5600 "
Sheep	1-6700 "	1-5000 "	1-6667 "
Goat	1-8100 "	1-6100 "	1-8133 "

A similar change in the form and size of many of the red blood-cells takes place when the blood dries slowly, so that, in preparing a blood-stain for microscopic examination, that portion of the stain should be selected that has dried most quickly, since there it would be expected to find a larger proportion of cells that have preserved their normal shape and size than in the center of the clot, where the drying has taken place more slowly.

When blood is treated with saline solutions of greater density than blood-serum, the red cells become shrunken or shriveled and the edges irregular, or what is termed "crenated," but they do not lose their density and color. The red blood-cells are generally destroyed by acids and alkalis. Thus, while there are many agencies that may cause a diminution in the size of the red blood-cells, as yet none is known outside the living body that will cause an increase in their size or diameter. The influence of disease upon the size and form of the red blood-cells will be referred to later.

The size of the red blood-cells differs considerably in different animals, and to a certain extent within narrow limits in the same animal. As a rule, in animals having nucleated red blood-cells, the cells are

[1] "Comparative Studies of Mammalian Blood," *Jour. Med. and Surg.*, July, 1888, p. 254.

much larger than in those having non-nucleated cells, as, for instance, in one of the Louisiana reptiles (*Amphiuma*), the red cell is about $\frac{1}{350}$ inch in its long diameter, large enough to be seen by the unaided eye. Of the mammals, as will be seen by the table below, the elephant has the largest red blood-cells, the average size being $\frac{1}{2745}$ inch. In medicolegal cases generally the question only arises as to the distinction between the blood of man and that of the domestic animals in the case of mammalian blood-stains. In these the red blood-cells vary in their average diameter from about $\frac{1}{6000}$ to $\frac{1}{3000}$ inch. As will be seen from the table, the authorities differ but slightly with reference to the average diameter of the human red blood-cell. All agree that it is between $\frac{1}{3300}$ and $\frac{1}{3200}$ inch. Gulliver and Formad place the average at $\frac{1}{3200}$ inch; Professor Wormley at $\frac{1}{3250}$ inch; the French Committee appointed by the Medicolegal Society of France in 1873, at $\frac{1}{3257}$ inch; Masson gives the same diameter, and Professor Carl Schmidt,[1] at $\frac{1}{3300}$

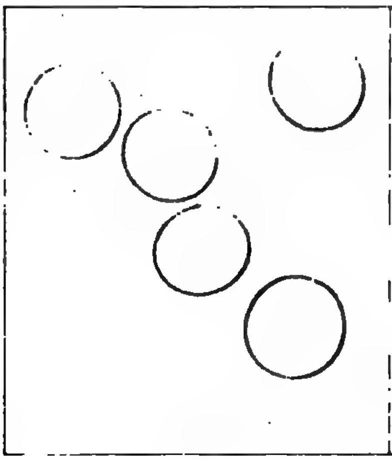


FIG. 82.—Human (× 2500).

FIG. 83.—Dog (× 2500).

inch. Professor Formad states, as the result of a large series of measurements, that 90 per cent. of the red blood-cells will measure between $\frac{1}{3300}$ and $\frac{1}{3100}$ inch, only 10 per cent., therefore, falling outside of these limits, which he considers the normal. The smallest red blood-cell is $\frac{1}{3800}$ and the largest $\frac{1}{2900}$ inch. Hayem[2] states that in every 100 red blood-cells about 12 will be larger than the average and about 12 smaller than the average.

The dog, pig, horse, ox, cow, cat, sheep, and goat are the only domestic mammals whose blood is liable to be of importance in medicolegal cases with reference to its distinction from human blood. Of these, the blood of the dog approaches most nearly in size to human blood. Figs. 82 and 83 show the red cells of man and dog respectively, magnified 2500 diameters. The average diameter of the red blood-cell

[1] "Die Diagnostic verdachtiger Flecke in Criminalfallen," Mitau and Leipsic, 1848.

[2] *Du Sang*, Paris, 1889.

in dog's blood is about $\frac{1}{3550}$ inch, while that of all the other animals mentioned is less than $\frac{1}{4000}$ inch; the red cells of the ox, pig, horse, and cat vary from $\frac{1}{4400}$ to $\frac{1}{4200}$ inch; the red blood-cell of the sheep is about $\frac{1}{5000}$, and that of the goat still smaller—less than $\frac{1}{6000}$ inch. The size of the goat's blood-cell is, therefore, approximately one-half that of the human.

The following table gives the average measurements as reported by various hematologists. The older authorities, Professors Gulliver and Carl Schmidt, obtained their results with low magnifying powers of the microscope, and agree astonishingly with the results obtained by recent authorities using higher powers and much more nearly perfect instruments. The figures given are in vulgar fractions of an inch, which is the scale almost universally used in this country, and which it is necessary to use in court, since the average juryman knows nothing about the metric scale. Where the original measurements were given in thousandths of a millimeter I have reduced them to vulgar fractions of an inch.[1] The first two columns of the table are taken from the appendix to the second edition of Professor Wormley's *Micro-Chemistry of Poisons*, p. 733 *et seq.*

AVERAGE SIZE OF THE RED BLOOD-CELLS.

MAMMALS.	Gulliver.	Wormley.	Formad.	Richardson.	C. Schmidt, 1848.	French Medicolegal Society, 1873.	Masson, 1885.	Dragendorff.	Woodward.
Man	1-3200	1-3250	1-3200	1-3224	1-3330	1-3257	1-3256	1-3300	1-3092
Monkey	1-3412	1-3382	.	1-3395					
Opossum	1-3557	1-3145							
Guinea pig	1-3538	1-3223	1-3400	. . .	. . .	. . .	1-3300	. . .	1-3213
Kangaroo	1-3440	1-3410							
Musk-rat	1-3550	1-3282							
Dog	1-3532	1-3561	1-3580	1-3542	1-3630	1-3479	1-3577	1-3628	1-3246
Rabbit	1-3607	1-3653	1-3662	. . .	1-3968	1-3681	1-3628	1-3968	
Rat	1-3754	1-3652	. . .	. . .	1-3968				
Mouse	1-3814	1-3743	.	.	1-4166				
Pig	1-4230	1-4268	1-4250	1-4230	1-4098	1-4233	1-4098	1-4098	
Ox	1-4267	1-4219	1-4200	1-4267	1-4385	1-4535	1-4233	1-4385	
Horse	1-4600	1-4243	1-4310	. . .	1-4456	1-4535			
Cat	1-4404	1-4372	. . .	.	1-4535	1-3907	1-4456	1-4535	
Elk	1-3938	1-4384							
Buffalo	1-4586	1-4351							
Wolf (prairie)	1-3600	1-3422	1-3450						
Bear (black)	1-3693	1-3656							
Hyena	1-3735	1-3641							
Squirrel (red)	1-4000	1-4110							
Raccoon	1-3850	1-4084							
Elephant	1-2745	1-2738							
Leopard	1-4319	1-4390							
Hippopotamus	1-3429	1-3560							
Rhinoceros	1-3765	1-3649							
Whale	1-3099	. .	. . .	1-3090					
Tapir	1-4000	1-4175							
Lion	1-4322	1-4143							
Ocelot	1-4220	1-3885							
Mule	. .	1-3760							
Ass	1-4000	1-3620							
Ground-squirrel	. .	1-4200							
Bat	1-4175	1-3966							
Sheep	1-5300	1-4912	1-5000	1-5300	1-5649	1-5080	.	1-5649	

[1] A thousandth of a millimeter is equal to 0.00003937 of an inch. It is often called a micromillimeter, or micron, and is represented by the Greek letter μ. These terms are frequently used in connection with the measurement of microscopic objects.

AVERAGE SIZE OF THE RED BLOOD-CELLS (*Continued*).

Mammals.		Gulliver.	Wormley.	Formad.
Ibex		....	1-6445	
Goat		1-6366	1-6189	1-6100
Sloth		1-2865		
Platypus (duck-billed)		1-3000		
Capybara		1-3190	1-3164	
Seal		1-3281		
Woodchuck		1 3484		
Musk-deer		1-12325		
Beaver		1-3325		
Porcupine		1-3369		
Llama	Long diameter	1-3361	1-3201	
	Short "	1-6229	1-6408	
Camel	Long "	1-3123	1-3331	
	Short "	1-5876	1-5280	
Birds.				
Chicken	Long diameter	1-2102	1-2080	
	Short "	1-3436	1-3483	
Turkey	Long "	1-2045	1-1894	
	Short "	1-3598	1-3444	
Duck	Long "	1-1937	1-1955	
	Short "	1-3424	1-3504	
Pigeon	Long "	1-1973	1-1892	
	Short "	1-3643	1-3804	
Goose	Long "	1-1836		
	Short "	1-3839		
Quail	Long "	1-2347		
	Short "	1-3470		
Dove	Long "	1-2005		
	Short "	1-3369		
Sparrow	Long "	1-2140		
	Short "	1-3500		
Owl	Long "	1-1763		
	Short "	1-4076		

Reptiles.		Gulliver.	Wormley.
Tortoise (land)	Long Diameter	1-1252	1-1250
	Short "	1-2216	1-2200
Turtle (green)	Long "	1-1231	
	Short "	1-1842	
Boa Constrictor	Long "	1-1440	1-1245
	Short "	1-2400	1-2538
Viper	Long "	1-1274	
	Short "	1-1800	
Lizard	Long "	1-1555	
	Short "	1-2743	
Batrachians.			
Frog	Long diameter	1-1108	1-1089
	Short "	1-1821	1-1801
Toad	Long "	1-1043	
	Short "	1-2000	
Triton	Long "	1-848	
	Short "	1-1280	
Proteus	Long "	1-400	
	Short "	1-727	
Amphiuma tridactylum	Long diam.	1-363	1-358
	Short "	1-615	1-622
Fishes.			
Trout	Long diameter	1-1524	
	Short "	1-2460	
Perch	Long "	1-2099	
	Short "	1-2824	
Pike	Long "	1-2000	
	Short "	1-3555	
Eel	Long "	1-1745	
	Short "	1-2842	
Lamprey (Circular)		1-2134	
Lamprey (Diam. of Nucleus)		1-6400	

Method of Examination.—In determining the size of the normal red blood-cells of man and different animals two methods have been adopted: One consists in preparing a thin layer of fresh blood and measuring the red cells while still suspended in the serum, and the other in preparing a very thin layer of dried blood and measuring those cells that have retained their normal form.

In making a layer of fresh blood sufficiently thin for this purpose care must be taken that the slides and cover-glasses should be perfectly clean and absolutely free from any grease or dust. To secure this, they should first be washed with water to which a little alkali has been added, then placed in a covered dish and covered with alcohol until they are to be used. When about to be used, the glasses should be removed from the alcohol and wiped dry with a clean linen cloth. A very small drop of blood is then placed upon the slide by touching the slide to the drop as it issues from a wound made by pricking the lobe of the ear or the tip of the finger with a sharp-pointed sterilized needle or lancet. The lobe of the ear or tip of the finger should be made previously sterile by washing with water and afterward with alcohol; the first drop of blood that issues should be wiped off, and the second or third drop touched with the glass slide. The drop on the slide should be immediately covered with a perfectly clean cover-glass, when the blood will spread out under the cover-glass, if the drop has not been too large, in so thin a

bringing out the red blood-cells to their normal shape and of freeing them from extraneous masses so that their edges may be brought plainly into view is very different. A minute fragment of the dried blood-stain must be removed with the point of a knife or a pair of sharp-pointed scissors, transferred to a glass slide, and moistened with some liquid menstruum that will serve to soften and gradually disintegrate the clot, but will not destroy the blood-cells. Various menstrua have been proposed, some authorities preferring alkaline fluids, others acid ones, and others neutral solutions.

Virchow's liquid consists of a 30 or 33 per cent. solution of caustic potash. This is the solution that was preferred by the late Professor Formad. Roussin recommended a mixture of 3 parts of glycerin, 1 part of sulphuric acid, and water to a specific gravity of 1028. Ranvier's serum was made by dissolving 2 grams of potassium iodid in 100 grams of water, and saturating this solution with iodin. Vibert used a solution of ½ part of corrosive sublimate, 2 parts of common salt, and 100 parts of water. Paccini's fluid was made by dissolving in a mixture of 300 parts of water and 100 of glycerin 2 parts of common salt and 1 part of corrosive sublimate. The late Dr. Richardson used a 0.75 per cent. salt solution, first washing out most of the blood coloring-matter, and then staining the cells with a solution of fuchsin. The late Professor Wormley recommended simply a small quantity of distilled water, adding approximately the amount originally present in the dried specimen under examination. If the stain was a very old one, so as to require long soaking in the menstruum, he recommended a dilute solution of glycerin, one having a specific gravity of 1030, and in cases of very old stains, which disintegrate with difficulty, he advised the addition of a little cautic potash to the glycerin or the water. The writer has obtained from a solution of potassium acetate of a specific gravity of 1030 very satisfactory results. The addition of a little formalin to this solution improves it by preventing the formation of any fungous growth.

Care must be taken in selecting portions of a dried stain for microscopic examination and measurements to take the dried fragments from some portion of the stain that has dried quickly. If the specimen be taken from the center of a mass of dried blood which has required several hours to dry, the red cells will have taken up a sufficient amount of water to render them spherical, and, therefore, unfit for measurement. The particle of stain thus selected should be moistened with whatever menstruum the observer prefers, covered with a thin covering-glass, and sealed with cement to prevent the evaporation of the fluid. If the blood has dried in a very thin layer upon some non-absorbent surface, the thin film may be scraped from the surface with a sharp knife on to a glass slide, and the cells fixed by heating them to 120° C. (248° F.) for fifteen or twenty minutes, or by moistening them with a mixture of equal parts of absolute alcohol and ether before treating them with the fluid. In this way the writer has often succeeded in obtaining a larger proportion of perfectly formed cells than if the heat had not been applied. The slide should be examined from time to time with a low power, and

as soon as cells of the normal form are seen, they may be measured with the high powers. It will be found that the dried blood in some stains disintegrates much more slowly than in others, and, other things being equal, the older the stain, the longer the time required. A stain a few days old disintegrates rapidly, so that measurements may be made as soon as the preparation is completed. Distorted and swollen red cells must not be measured.

More satisfactory results are obtained in cases in which the blood has fallen upon a non-absorbent surface. When possible, the examiner should attempt to select a dried crust as thin as possible. If the blood has fallen upon a piece of linen or cotton cloth in small quantity, so that it has penetrated the fiber of the cloth immediately, as a general rule only unsatisfactory results can be obtained, because the cells adhere tightly to the individual fibers, which, while moist, are somewhat swollen and contract upon drying. By this contraction the adherent blood-cells frequently become so distorted as to become unfit for measurement. In such cases a satisfactory result can usually be obtained only when, for some reason or other, complete absorption of the fresh blood has been prevented, as, for instance, if the cloth happened to be more or less starched or if the drop of the fresh blood was a little too large to be completely absorbed at once. Nucleated blood-cells can, however, readily be seen under these circumstances. The difficulty of discrimination occurs only in the case of mammalian blood-stains.

Blood-stains that have been subjected after drying to long-continued action of moisture are generally unsuitable for microscopic measurement, since the red cells absorb a sufficient amount of water to render them abnormal. If the stain has been formed upon a non-absorbent surface and has been kept dry, a sufficient number of the red cells will preserve their normal form, so as to be suitable for microscopic measurement, for an indefinite number of years. Blood-stains upon iron can be satisfactorily examined only if the iron has been kept completely dry, so that rusting of the iron has not taken place. If, however, the iron has rusted at the point where stained, not only the blood-cells may be totally destroyed, but the blood-pigment may also be decomposed.

Blood-stains are frequently submitted to the expert in which an attempt has been made to wash off the blood with water. Thorough washing will, of course, remove all traces of blood from cloth, but it frequently happens that a hasty attempt to remove the blood-stain, particularly from cloth having a rough surface, will leave a sufficient number of particles of unchanged blood to render a satisfactory examination and measurement possible. If a blood-stain on cotton or linen cloth is treated with water, the blood-pigment is taken up by the water and carried along with it, so that after drying the whole portion of the cloth that has been wet will show a yellowish stain, but the color will be deeper and browner at the edge than in the center. This is called a diffused or a washed stain, the edges of which, after drying, may be of a deep-brown color, while an inch or two from the edge the color is of

a much lighter tint. Sea-water removes the blood from a dried stain much more slowly than fresh water.

In the examination of blood-stains great care should be taken to note every peculiarity of each individual stain. As has been mentioned above, the form of the stain, together with the distribution of the blood in it, may show from which direction the blood making the stain started. Note should be made as to whether the stain is a spatter or a smooch, or whether it has the appearance of having been wiped or washed. Careful examination should also be made to determine the presence in the stain of any foreign substances, such as hairs, pieces of tissue, muscular fibers, or adipose tissue, bits of bone, etc. It may happen that the presence of such substances will throw light upon the nature of the blood making the stain. For instance, any hair found in a blood-stain may resemble that of the victim and be unlike that of the accused, or in case the tissues have been much bruised by a blunt instrument, or perhaps bones fractured and crushed, it is not uncommon to find small particles of tissue, or even a minute fragment of bone, mixed with the blood. Sometimes these foreign substances may be detected by gross inspection; at other times the fragments may be so minute that their presence can be detected only by the microscopic examination.

The following table, taken from Professor Wormley's article, gives the results obtained by him in the examination of dried blood-stains made by different kinds of mammalian blood, the nature of the blood in some instances being unknown to him at the time of the examination:

TABLE OF PROFESSOR THEODORE G. WORMLEY'S MEASUREMENTS.
EXAMINATION OF OLD BLOOD-STAINS.

ANIMAL.	Age of stain.	Remarks.	Average.	Fresh blood.
(1) Human . .	2 months old	Stain; unknown . . .	1–3358 inch	1–3250 inch
(2) Human . .	2½ "	Stain	1–3236 "	1–3250 "
(3) Human . .	3 "	Stain	1–3384 "	1–3250 "
(4) Human . .	19 "	Clot	1–3290 "	1–3250 "
(5) Elephant .	13 "	Clot	1–2849 "	1–2738 "
(6) Dog . . .	4 "	Trace of stain; unknown	1–3626 "	1–3561 "
(7) Rabbit . .	18 "	Clot	1–3683 "	1–3653 "
(8) Ox	16 "	Stain	1–4544 "	1–4219 "
(9) Ox	32 "	Stain; unknown . . .	1–4495 "	1–4219 "
(10) Ox	4½ years old	Clot	1–4535 "	1–4219 "
(11) Buffalo . .	18 months old	Clot	1–4312 "	1–4351 "
(12) Goat . . .	17 "	Stain	1–5897 "	1–6189 "
(13) Ibex . . .	18 "	Clot	1–6578 "	1–6445 "

Influence of Disease on the Red Blood-cells.—It is well known that in certain diseases the red blood-cells may be altered, both in size and in form, and also the relative proportion between the red and the white blood-cells. Some diseases will diminish their size, as high fever, diphtheria, and septicemia. In pernicious anemia we find some of the red blood-cells of very large size; these are called megalocytes. The diameter of these may be almost double the diameter of the normal red blood-cells. In this disease we may also find many red cells much smaller than normal, even to one-half the normal diameter, called mi-

axis Erxl, India), dog, cat, pig, bat (*Plecotus auritus*), pigeon, chicken, pheasant, swan (*Cygnus olor*), duck, chaffinch (*Fringilla coelebs*), cross-bill (*Nucifraga caryocatactes*), rook (*Corvus frugilegus*), swallow (*Hirundo urbica*), corn-crake (*Crex pratensis*), frog (*Rana temporaria*), newt (*Molge cristata*), snake (*Tropidonotus natrix*). The serum of the rabbit that was prepared with the sheep's blood gave a slight reaction with the blood of the gazelle and axis deer, and there was very slight clouding in the blood of the ox, squirrel, and swan. The serum of the rabbit treated with ox-blood gave a distinct reaction with the blood of the gazelle and axis deer, and slight cloudiness in the blood of the sheep, gnu, squirrel, and swan. The serum of rabbit prepared with human blood gave a slight reaction with the blood of the four kinds of monkeys tested. He found that it gave a very faint cloudiness in solutions of the horse, ox, and sheep. The serum of the humanized rabbit gave positive reactions with diluted human serum, pleuritic exudation, both fresh and putrid, with blood that had undergone putrefaction for two months, with serum from a blister, and a slight reaction with both nasal and lacrimal secretions.

Later, Dr. Nuttall published further tests of more than 500 specimens of blood from different sources, the results of which have been corroborative of the above.[1]

The method of humanizing a rabbit is to inject into the peritoneal cavity about 10 c.c. of human blood-serum at intervals of two or three days until the rabbit has received 6 or 8 injections. The rabbit should be allowed to rest about a week after the last injection. The blood-serum for performing the test may then be obtained either by killing the animal by bleeding it from one of the large vessels, or without killing the animal by removing a little blood from one of the large veins of the ear. The blood thus collected should be placed in a cool place and allowed to coagulate. The serum that separates from the clot may be used for performing the test.

The blood to be tested should be prepared as follows: If it be fresh blood, it should be diluted about 1 : 100 with normal salt solution. Thus diluted, it should have a light-pink color, and this diluted solution, if not perfectly clear, should be allowed to settle until it is clear, and the clear, supernatant fluid decanted into another test-tube. If the blood to be tested is a dry blood-stain, a little of it may be scraped off with the point of a knife on to a watch-glass, if the stain be upon some hard surface so that the blood has not penetrated into the substance of the material. If the blood-stain be upon cloth into which the blood has soaked, it is necessary to cut out a few threads and transfer them to a watch-glass. These fragments of dried blood should then be treated with two or three drops of distilled water until the soluble portion of the dried blood has been dissolved. The clear solution should then be transferred carefully to a very narrow test-tube, and to this should be added an equal volume of double normal salt solution. The clear fluid thus obtained either by diluting fresh blood or by dissolving the blood-

[1] *Brit. Med. Jour.*, April 5, 1902, p. 825.

serum from the dried blood-stain is then tested by adding to it a few drops of the serum obtained as above described from the humanized rabbit. If the solutions contain human blood-serum, there will occur an immediate cloudiness that gradually increases, so that there is a distinct precipitation within one-half hour after the addition of the serum from the humanized rabbit. It is better often to allow the antiserum to flow down the side of the test-tube, so as to form a separate layer under the solution to be tested, in the same way that we add nitric acid to urine in testing for albumin. In this case the cloudiness and precipitate may be seen very distinctly in the zone at the point of contact of the two fluids. The precipitate occurs best when the mixture is kept at a temperature of about 37° C.

Ewing[1] has shown that, in applying the serum-test, it is important that the solution of the blood under examination be not too strong; if it is too concentrated, the blood of some of the lower animals may react to a greater or less degree. At a dilution, however, of at least 1 to 50 the blood of lower animals fails to give a reaction, and the test becomes a specific one for human blood. Dilution of the serum is even better than dilution of the blood.

It has been found that the antiserum obtained from the humanized rabbit can be kept in dried form by soaking filter-paper or blotting-paper with it, and allowing it to dry. In this way it is said to preserve its activity for forty-two days, more or less (Dr. Nuttall). When it is desired to use this for a test it may be dissolved from the filter-paper by means of a little normal salt solution and filtered if necessary. This clear solution, added to the diluted blood or solution from a dried human blood-stain, will give the cloudiness and precipitation the same as the original antiserum obtained from the humanized rabbit.[2]

CONCLUSIONS.

By far the majority of hematologists who have investigated this subject during the past twenty years agree that discrimination can often be made in the case of dried blood-stains between human blood and that of the domestic animals.

If upon examination of a dried blood-stain by the microscope we find that the red cells are oval and nucleated, we may at once state that the blood of the stain did not originate from a mammal, but from one of the oviparous animals.

If the red blood-cells seen are biconcave dises and have an average diameter between $\frac{1}{3300}$ and $\frac{1}{3100}$ inch, we may say that the blood of that stain did not originate from any mammal the average diameter of whose red blood-cells is less than $\frac{1}{4000}$ inch, which excludes the blood of most of the domestic animals—ox, horse, pig, sheep, cat, and goat.

[1] *Med. Record*, February 28, 1903, p. 356.

[2] For further information concerning the serum-test for blood consult "The Biological or Precipitin Test for Blood Considered Mainly from its Medico-Legal Aspect," by G. S. Graham-Smith and F. Sanger, *Jour. Hygiene*, April, 1893, p. 258, and July, p. 354.

The blood of the dog, guinea-pig, and rabbit has red cells whose average diameter is greater than $\frac{1}{4000}$ inch, and many hematologists do not consider that we are warranted, from the size of the red blood-cells alone, in expressing a positive opinion as between human blood and that of one of these animals, which may be included in the list of domestic animals. All that the expert is required to state is that these measurements ($\frac{1}{3300}$ to $\frac{1}{3100}$ inch) are *consistent* with the human origin of the blood in question. The burden of proof always lies with the defendant to state that it is not of human origin.

The serum-test is the most delicate one thus far discovered for differentiating between the different kinds of blood, and has been shown to be applicable to both fresh and decomposed blood and to dried blood-stains.

OTHER STAINS CONTAINING BLOOD.

Diagnosis of these can be made only by finding, on microscopic examination, other formed elements mixed with the blood-cells, such as epithelial cells of various kinds, pus, dried mucus, spermatozoa, etc.

Menstrual Stains.—The possibility of determining whether or not a blood-stain was caused by menstrual blood depends largely upon the amount of hemorrhage. This, as is well known, differs with different women, and also in the same individual at different times. If the flow be scanty, as at the very beginning or end of the menstrual period, the menstrual blood will have mixed with it a large number of epithelial cells coming from the vagina. If, however, the flow is very abundant, a stain may be made by menstrual blood, particularly a small stain, which does not contain any of the vaginal cells.

The vaginal cells are large polygonal, squamous, epithelial cells, somewhat similar to those obtained from the mucous membrane of the mouth, but having on the average a somewhat larger nucleus. Sometimes we see a vaginal cell with two nuclei. These cells may be exfoliated singly or in small patches containing several layers of cells. If the vagina be inflamed, as in leukorrhea and gonorrhea, these cells will be exfoliated from the membrane in much larger number, and will be mixed with pus-corpuscles. Possibly a cylindric or ciliated cell from the lining membrane of the uterus may be seen mixed with the blood.

If, therefore, we find mixed with the red blood-cells a number of vaginal epithelial cells, we may state that the stain in question is consistent with its having been made by menstrual blood. If, however, we do not find any vaginal cells mixed with the blood, we are not warranted in concluding that the stain was not made by menstrual blood.

The location of the stains, as upon bedding or underclothing, may possibly be of service in deciding the question as to the menstrual origin of the blood-stain in question. In uncleanly women these stains are said to be found more frequently on the back part of an undergarment than on the front.

Nasal Blood-stains.—The detection of nasal blood-stains depends upon precisely the same principle as that of menstrual stains, namely,

the finding of various morphologic elements coming from the mucous membrane of the nose mixed with the blood. These morphologic elements are coagulated mucus and cylindric or ciliated epithelial cells coming from various portions of the Schneiderian mucous membrane. If there is but little hemorrhage and the stain be made by forcibly blowing the nose, the blood will be mixed with more or less mucus and cells, and when such a stain becomes dry, it presents a very different appearance from an ordinary blood-stain; it is paler and more bulky than a blood-stain, and, after being moistened, if there is much dried mucus present, it will swell up and have a more or less elastic feel.

If the hemorrhage from the nose has been profuse, a large portion of the blood may not have mixed with it any of the mucous secretion or any of the cells, so that stains may be made from blood coming from the nose, which do not differ in any way from a pure blood-stain. In such cases the location of the stain in question may show whether or not it could have been made by blood coming from the nose. For instance, in a recent case investigated by the writer, in which the accused alleged that the blood upon his clothing was due to nose-bleed, a horizontal spatter of blood was found between the folds of a turn-down collar, in such a position that it could not have come from the nose while the collar was around the neck.

As in the case of menstrual stains, therefore, we can only state that a stain in which we have found coagulated mucus and cells mixed with the blood is consistent with its having originated from the nose. If we do not find such an admixture, we cannot say that the stain was not caused by nose-bleed unless it is so located that it would be impossible for the blood coming from the nose to have gotten into that position.

MEDICOLEGAL EXAMINATION OF SEMINAL STAINS.

THE recognition of a seminal stain upon clothing or other substance is, naturally, of great medicolegal importance in cases of alleged rape or sodomy. The variety of substances liable to be submitted to the expert for examination for seminal stains is not so great as for blood-stains. The articles usually brought for examination are pieces of bed-clothing or underclothing, and sometimes other articles of wearing apparel; in some cases scrapings from the skin or mucous membrane of the alleged victim, such as dried masses taken from the neighborhood of the genitals or tightly adherent to the hair, or scrapings of mucus from the vagina. Rarely are extraneous substances, such as pieces of wood, metallic instruments, or bits of leaves or earth submitted for examinations of this kind. Seminal stains, like blood-stains, may be either simple or complex—that is, they may consist of pure dried seminal fluid or of seminal fluid mixed with other fluids or secretions, such as blood, discharges from vagina, intestines, or other membranes.

Seminal fluid varies somewhat in its consistence according to circumstances, and in the same individual at different times. Its density may vary from 1027 to 1037; it is more or less viscid and feebly alkaline; usually it has a grayish, more or less opalescent appearance, and in rare instances it may have a reddish tint, even when not mixed with blood, and more particularly is this the case in old age. When fresh, it has a peculiar odor that is entirely lost upon drying. Formerly considerable importance was attached to the odor as a medicolegal test, but at the present time this test is never employed in dried stains, as many other substances have been found that, particularly when heated, yield an odor closely resembling that of heated seminal fluid.

The appearance of a dried seminal stain varies according to circumstances, and particularly according to the nature of the surface on which the stain is made. If found upon a non-absorbent surface, such as a piece of wood or iron, or upon heavy woolen cloth or velvet, it dries, forming a grayish scale upon the surface of the fabric, from which a portion can very easily be removed with the point of a knife-blade or a needle for microscopic examination. If cotton or linen cloth has been starched, so as to render it comparatively non-absorbent, a seminal stain will have the same scaly appearance. The color of the stain may be simply grayish or have a very faint yellowish tint, and in rare instances the stain may have a reddish tint not uncommon with the seminal fluid of old men. On unstarched cotton, linen, or any very absorbent fabric

a seminal stain is very difficult to see, especially if the stain be a small one. When a drop of the fluid falls upon such a fabric, it is immediately absorbed, and the fluid portion extends for quite a long distance, forming, when dry, a nearly colorless stain having a very irregular outline. In some instances scarcely any change in color is produced upon the surface of the cloth, but if the cloth be held up to the light and viewed by transmitted light, the stained portion will be somewhat translucent, the meshes of the cloth being more or less filled with the dried material. The stained portion will also be found to have a stiffer feel than the unstained portion of the cloth, as is the case when such a fabric is stained with blood or any albuminous fluid. If the seminal fluid forming a stain is mixed with other secretions, the appearance of the dried stain would, of course, be modified accordingly.

The location of seminal stains may vary greatly with different circumstances. In cases of alleged rape they are found most frequently upon the underclothing of the victim or upon the clothing of the accused. In cases of rape of young girls the stains are almost invariably found upon the posterior flap of the undergarment or upon the drawers or shirt. On the undergarments of men the stains are almost invariably upon the anterior portion.

The Florence Test.—Seminal stains are diagnosticated with absolute certainty only by the recognition of the characteristic morphologic elements, the spermatozoa, by microscopic examination. The seminal fluid consists of an admixture of the secretion of several glands, the fluid portion being a solution of various organic and inorganic substances, being especially rich in phosphates, and the undissolved portion consisting of the characteristic spermatozoa and numerous cells coming from the mucous membranes lining the different portions of the seminal tract. The seminal fluid does not contain serum-albumin, but it does contain other albuminoid substances. Of the various organic substances that appear to be almost constantly present are nuclein, lecithin, cholesterin, hypoxanthin, and fatty matters rich in phosphorus; cerebrin, guanin, and sarcin have also been found. A crystalline substance called spermin was first isolated from seminal fluid by Schreiner in 1878. It has the formula C_2H_5N, and gives all the general reactions of an alkaloid. It is soluble in water and absolute alcohol, but very slightly soluble in ether; its solutions have an alkaline reaction. Spermin is not peculiar to the seminal fluid of man, but has been found in that of some animals, also in sputum, blood, and some of the animal tissues. Dr. Florence,[1] of Lyons, claims to have discovered an alkaloidal body that he finds only in the seminal fluid of man, and that gives a characteristic crystalline precipitate with a concentrated solution of iodin in potassium iodid. Dr. Florence has not been able to obtain these crystals with the seminal fluid of any other animal, nor from any other fluid or tissue. This substance Dr. Florence calls virispermin. This test is one of extreme delicacy, a single fibril of cloth upon which is a dried seminal stain sufficing to yield numerous crystals when the test is properly performed.

[1] *Du Sperme et des Taches de Sperme en Médecine Légale*, 1897.

The reagent, ioduretted potassium iodid, is prepared in the following manner:

Potassium iodid	1.65 gm.
Iodin	2.54 "
Distilled water	30.00 "

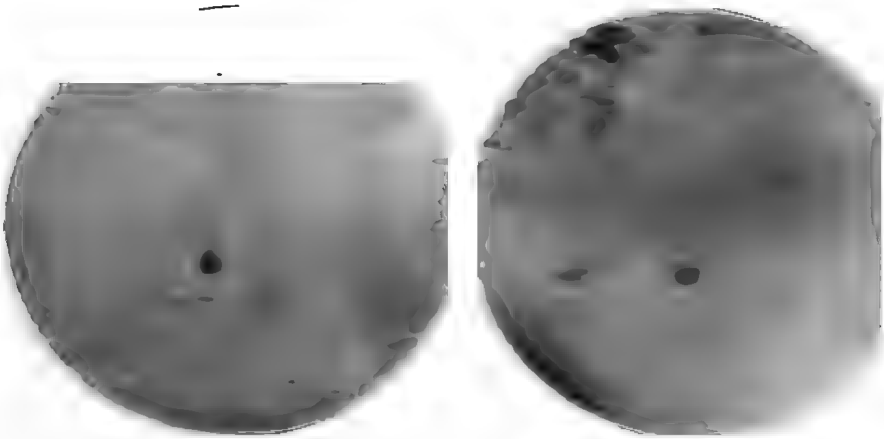
These proportions correspond to the formula KI_3. The iodin dissolves very quickly in the solution of potassium iodid, and it is only necessary to mix the material together in a glass-stoppered bottle and allow it to stand for a short time, when the reagent will be ready to use.

The test is performed in the following manner: A very minute fragment of the stained fabric is carefully removed by means of fine-pointed forceps and sharp-pointed scissors, transferred to a glass slide, treated

FIG. 84.—Seminal crystals (small) (× 750).

with a small drop of distilled water, allowed to soak for a minute or two, a minute drop of the iodin reagent added to it in such a way that the two drops of fluid come in contact by their edge, and immediately covered with a covering-glass. If the suspected stain contained any dried seminal fluid, the examination with the microscope will show a very large number of brown crystals that resemble in appearance the hemin crystals obtained by performing Teichmann's test with blood. The accompanying illustrations (Figs. 84, 85, and 86) show these crystals, which were obtained from a single thread $\frac{1}{8}$ of an inch long cut from the stained portion of a pair of child's drawers in a case of attempted rape, the stain being three years and four months old at the time the test was made.

If the stain examined be one upon a non-absorbent surface, like a piece of wood, it is sufficient to remove a very minute fragment of the



more or less pear-shaped. In the examination of stained spermatozoa it will be seen that the anterior third of the head is less dense than the posterior two-thirds, and is stained by the various color reagents less deeply. This is well shown by the accompanying cut (Fig. 87), taken from a dried and stained specimen. Fig. 88 is of a spermatozoön isolated from the seminal stain three years and four months old on a child's drawers.

The spermatozoa are very stable and do not become readily destroyed, so that they may be detected in stains many years old if the stain has not been subjected to too much washing or exposure, as shown by Fig. 88. These bodies are, however, after they become dry, very brittle, so that the tail is very easily separated from the head, and it is for this reason that it is so difficult to obtain many perfect spermatozoa in the examination of old dried seminal stains.

For the positive recognition of spermatozoa it is, in our opinion, necessary to find the perfect bodies with head and tail complete, since there are other bodies, such as certain spores, sometimes found in old stains, which very much resemble the heads alone, and there are numerous substances, such as bacteria or bits of the fibrils of cloth, which frequently resemble the tails alone. Therefore great care should be exercised in the handling of any fabric or other substances suspected to contain seminal stains which are to be submitted for medicolegal examination. They should not be subjected to any unnecessary handling or rubbing, and should be submitted to the expert for examination in as fresh a condition as possible.

The recognition of spermatozoa is very much facilitated by staining them with suitable staining solutions. Dr. Florence recommends as the best solution for this purpose a concentrated aqueous solution of crocein. The writer has not had the opportunity of trying this solution, but has found the ordinary solutions of methyl-green or eosin very useful for this purpose.

If the stain is a scaly one, such as is made by the drying of the fluid upon a non-absorbent surface, the detection of the spermatozoa is comparatively easy. A fragment of the stain, removed with the point of a knife, should be transferred to a glass slide, treated with a drop of distilled water, and allowed to soak for several hours under conditions that will prevent the rapid evaporation of the water. The scales should then be gently separated with fine needles, treated with a drop of the staining solution, covered with a cover-glass, and examined directly with the microscope. A power of from 500 to 700 diameters should be used. In some cases it is advisable to confirm the result by examining with an immersion lens. Sometimes a better preparation can be made by first soaking the particles of stain with water on a cover-glass for several hours, then pick to pieces, and allow to dry. When dry, pass the cover-glass three or four times through the lamp-flame, or heat the cover-glass in an air-bath to 115° C. (239° F.) for fifteen or twenty minutes, so as to fix the spermatozoa upon the glass, in the same way that the red and white blood-cells are fixed previous to being stained in the clinical examination of blood by the Ehrlich process. Then treat

the dry residue upon the cover-glass with the staining solution for from three to five minutes, wash off the superfluous stain, and dry. This preparation can be mounted dry, sealing the cover-glass with paraffin, or it can be mounted in Canada balsam, or can be mounted wet in water or any desirable menstruum.

If the stain is upon unstarched cotton or linen cloth, the detection of the spermatozoa is much more difficult. The spermatozoa are apt to cling to the fibrils so tenaciously that it is difficult to separate them without breaking the head from the tail. Care should be taken to select for the examination a thread of the cloth near the center of the stain, and the cloth should first be carefully examined with the lens in order to see, if possible, if one side contains more dried stain than the other. For instance, stains have been found that yielded an abundance of spermatozoa on one side of the cloth, while none could be found on the other, although the Florence reaction could be obtained on both sides. Also the fluid portion of the seminal fluid may be sucked along for quite a distance upon a piece of linen or cotton cloth, while the solid bodies, the spermatozoa and cells, may be limited to near the center of the stain; in this case the Florence reaction will be obtained from the edge of the stain as well as from the center, but the spermatozoa can be isolated only from the central portion of the stain.

The best method to prepare such a stain for microscopic examination is carefully to cut from near the center of the stain, with a pair of fine-pointed scissors, a few individual threads of cloth from $\frac{1}{16}$ to $\frac{1}{8}$ of an inch long, and treat each piece of thread upon a glass slide or cover-glass with a small drop of water, allowing it to soak at least two hours, the evaporation of the water being prevented. Some authorities think that the soaking should continue from twelve to twenty-four hours. Numerous preparations should be made, since it frequently happens, even in well-marked seminal stains, that careful search will fail to detect unbroken spermatozoa in one or more of the preparations. After sufficiently prolonged digestion the small fragment of thread should be very carefully separated into its individual fibrils by manipulating with sharp-pointed needles. It can then be stained and examined as described above in connection with the more scaly stains upon non-absorbent material.

Moist material, such as mucus obtained from the uterus or vagina, may be at once placed upon a glass slide, stained with one of the color reagents, and examined at once for the detection of spermatozoa.

According to Florence, the spermatozoa of none of the domestic animals have the pear-shaped profile that human spermatozoa have.

The Biologic Test.—Dr. C. G. Farnum [1] has proposed a biologic test for human semen that is based upon the same principles as the serum-test for blood. He prepares the rabbit by injecting into the peritoneal cavity from 5 to 10 c.c. of either semen or testicular emulsion at intervals of from two to six days, the rabbit receiving from 5 to 8 injections. The test is performed in a similar manner to the serum-test for blood.

[1] *Jour. Amer. Med. Assoc.*, December 28, 1901, p. 1721.

MEDICOLEGAL EXAMINATION OF HAIRS.

A NUMBER of questions present themselves in connection with the medicolegal examinations of hairs, among the most important of which are the following:

1. Is the material examined hair or some other fiber?
2. If hair, from what animal did it come?
3. If the hair is human, can it be identified as the product of a given individual?
4. If of human origin, from what part of the body did the hair come?
5. What was the age of the person from whom the hairs were derived?
6. How were the hairs removed from the body?

The first question is usually easily answered. It is true that to the naked eye various articles, chiefly vegetable fibers, may simulate hairs; but by the aid of the microscope all other bodies may be distinguished from them.

The distinctive features of hairs that are recognized by even low powers—200 to 300 diameters—are: 1. The shingled or terraced ("imbricated") surface. 2. The distinction between cortex and medulla. 3. The pigment-granules in the cortex. 4. The cells composing the medulla. 5. The transition in structure from shaft to root, and the peculiar sheath of the latter.

It is seldom that all these features are found in a single specimen submitted in a judicial inquiry; but, fortunately, positive identification can be made through the detection of several of them.

If needed, confirmation of the identity can be secured by submitting the supposed hair to certain chemical reagents; a strong mineral acid, heated, causes the disintegration of a hair into the component cells.

The bodies from which hairs must, by these features, be distinguished are cotton, linen, silk, wool, and vegetable fibers (Plate 13). Cotton is a flat, tape-like fiber with a tendency toward a spiral twist; linen is a jointed, bamboo-like fiber with transverse markings at irregular distances; silk is a cylindric fiber without markings, but strongly refractive. Wool, as a variety of hair, presents the imbricated surface characteristic of hairs: while in general cylindric, its diameter varies in different parts. Any of these fibers may, of course, present the colors imparted by the dyes used in the cloth.

The second question is from what animal the hairs have been separated. Fortunately, the query does not usually include the whole cata-

logue of hairy animals; else the solution of the problem might become practically impossible, because of the great similarity between individual hairs of different species. Practically the only animals requiring consideration are those which, living or dead, enter into human environment—the domesticated quadrupeds, the parasitic rodents, the animals whose skins are converted into clothing. The hairs of these represent different types, and can all be distinguished positively from human hair (Plates 13, 14).

The third question often presented to the microscopist in a legal inquiry is whether hairs admittedly human can be identified as the product of a given individual; whether they must have grown on the body of a certain person, and could not have been furnished by any other.

This question must be answered always and distinctly in the negative. While similarity in color and size with that of a given individual may be marked, there is nothing peculiarly characteristic in the hairs of one person to distinguish them from the hairs of many others of similar complexion. It may occasionally happen—as in one case examined by the writer—that the hairs in question present certain peculiarities of structure due to disease; the detection of similar abnormalities in hairs from the suspected source would naturally furnish a probability, but not a certainty, of a common origin. Aside from these necessarily rare cases, identification of the individual from whom given hairs are derived is dependent upon community of size, length, and color; and these are insufficient to warrant more than a probable conclusion.

A fourth question is, From what part of the human body are the hairs under examination derived? This question may often be answered definitely because of the following facts:

1. The long, soft hairs from the scalp and beard are distinguished both by their length and by their gradual tapering from root to point.

2. The short, thick, stiff hairs from the eyelashes and the eyebrows, while averaging almost the same thickness as scalp hairs at the root, taper rapidly toward the point.

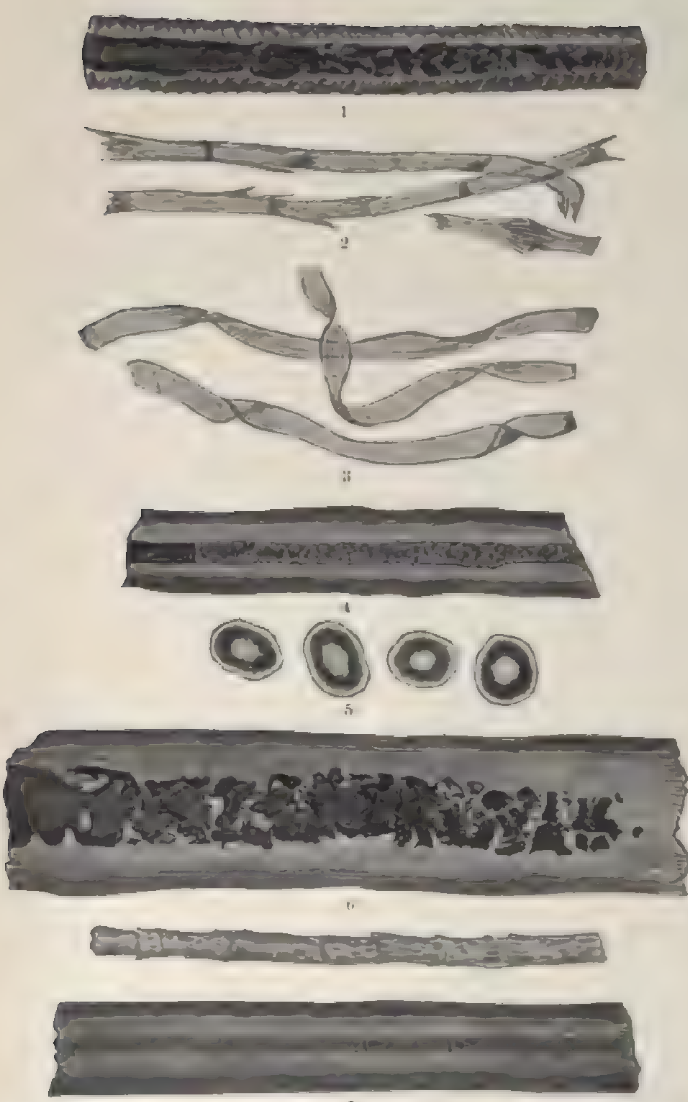
3. The short, slender, flexible hairs from the general surface of the body—the so-called lanugo or down—have, on the average, a much smaller diameter than other hairs, even than the equally short but thick hairs from the eyelashes. These downy hairs, moreover, frequently exhibit no pigment-granules in the cortex; and the medullary canal is apt to be relatively small and is frequently absent.

The average diameters of hairs from different parts of the body may be approximately stated as follows:

Scalp hairs, $\frac{1}{350}$ inch in the male, $\frac{1}{450}$ inch in the female; the variations in different persons may, however, be extreme—from $\frac{1}{1000}$ to $\frac{1}{200}$ inch, for example.

Hairs from the beard and mustache are usually the thickest of the body, ranging from $\frac{1}{250}$ to $\frac{1}{150}$ inch; they usually exhibit the greatest diameter on the chin and upper lip, shading off in diameter as they approach the scalp above and the neck below.

PLATE 13.



1, Sheep's wool (× 200). 2, Linen fibers (× 200). 3, Cotton fibers (× 200). 4, Hair from human head (× 200). 5, Transverse sections of hairs from human head (× 200). 6, Hair from human beard (× 200). 7, Hairs from back of hand (× 200).

Hairs from the eyebrows, lids, axillæ, and pubic region present about the same diameter at their roots as do those of the scalp; though here again great variations are found in different individuals.

The downy hairs from the general body surface are notably less in diameter than those in the so-called hairy parts of the body; they average from $\frac{1}{2000}$ to $\frac{1}{1000}$ inch in diameter near the root.

A fifth question sometimes propounded to the expert microscopist is, What was the age of the person from whom the hairs were derived? Here again his answer is restricted by serious limitations, and must be based upon the application, to the question at issue, of the following facts:

The downy hairs of the fetus and of the new-born infant contain no pigment and no medullary canal.

The hairs of children before puberty frequently have no medullary canal; they are relatively slender when compared with hairs of the same length and locality from adults.

A sixth query to which an answer may be sought through the microscope is, Were the hairs forcibly pulled out, shed from natural causes, or cut off?

This question can be answered, as a rule, decisively on the following facts: The root of a growing hair is concave, fitting over the convex papilla from which it grows. When forcibly separated from the head, the root end shows this concavity, as a rule; a bunch of hairs, most of which exhibit this feature, may with certainty be affirmed to have been forcibly pulled from the body. On the other hand, hairs which have fallen from natural causes exhibit little or no concavity at the root end.

A hair which has been cut from the head or other part naturally shows a plane surface, or something approximating this, where the cutting instrument severed the hair. The cut surface is sometimes uneven, because the different fibers composing the shaft have been severed at various levels.

The hairs growing upon the human body may be considered in three categories:

1. The long, soft hairs from the head.
2. The short, stiff, thick hairs from the eyelashes.
3. The short, slender, flexible hairs from the general surface of body and face—the so-called lanugo or down.

The long hairs from scalp and beard are naturally the most frequent subjects of microscopic examination for judicial purposes; in rarer cases the downy hairs, overlooked perhaps by naked-eye examination, have been magnified by the microscope into formidable witnesses of guilt. In one case in which the writer was engaged the presence of these diminutive hairs in blood-stains on a knife strongly confirmed the inference, from measurements of the blood-corpuscles, that the blood was of human origin.

In all cases where the identity of hairs is to be determined it is extremely desirable that several hairs be obtained for examination; because some of the structural peculiarities upon which the recognition

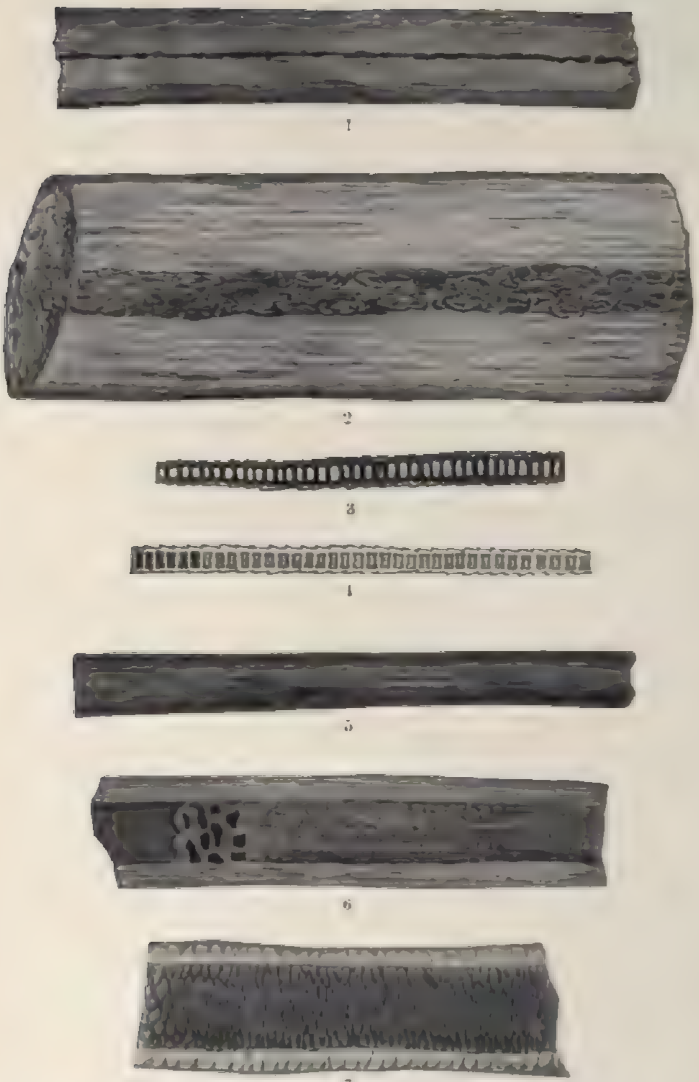
of the animal source depends may be absent in any single hair. Thus the medulla of human hairs—whose appearance and relative diameter to that of the entire shaft are important features—disappears in some sections of the hair, especially near the point; the pigment-granules also, whose size and distribution are characteristic of human hairs, may be nearly or quite lacking in individual specimens.

The domestic animals whose hairs approach most nearly in structure those from man are the dog and cow; yet the distinction is rarely difficult to one familiar with them all.

Thorough study of the structural peculiarities of hairs from all available sources must precede the attempt to identify given hairs in a judicial inquiry.

The usual method of preparing hairs for microscopic examination is to wash thoroughly with water, dry carefully, immerse in turpentine oil, and mount in Canada balsam.

PLATE 14.



1, Hair of ox (× 200). 2, Pig's bristle (× 200). 3, Cat's hair (× 200). 4, Mouse's hair (× 200). 5, Horse's hair (× 200). 6, Donkey's hair (× 200). 7, Goat's hair (× 200).

DEATH FROM POUNDED GLASS AND OTHER MECHANICAL IRRITANTS.

SHARP-CORNERED or pointed objects, especially particles of glass, are popularly supposed to be an efficient means of producing death, and have been considerably resorted to, more particularly by the insane, for the purpose of suicide; they have also occasionally been administered with homicidal intent.

Although such articles are frequently spoken of as poisons, they are not such in the true acceptation of the word.[1] They are practically insoluble, and do not, therefore, enter the circulation. Their effects are not produced after absorption, but are purely mechanical and local. They do not, consequently, strictly come under the head of poisons, but are included in the expression "other noxious things" of the statutes.

While any object with sharp points or angles is capable of producing gastro-intestinal irritation, the article of greatest interest from a medico-legal standpoint is pounded glass, and this, therefore, deserves chief attention.

Pounded glass is commonly looked upon as extremely dangerous when taken internally, and is often spoken of as an efficient "poison." There can be no question that the ingestion of particles of glass of a certain size may lead to such injury of the mucous membrane of the stomach and intestines as to cause death. A number of cases are on record of its fatal use, and in Russia death from its criminal administration is said not to be infrequent.[2] It is a favorite method of destroying certain of the lower animals, like dogs, rats, and mice, and its efficiency has led to its extensive use by the official rat-catchers of Manila[3] and of other Oriental cities in their war against the rodent propagators of the plague.

Still there can be no doubt that the dangers of broken glass and of other similar objects have been greatly overestimated. The results of experiments and the experience of professional glass-eaters indicate that they are far less dangerous than is commonly supposed. Glass suitably powdered has even been proposed as a remedial agent, Johns[4] claiming that it is an efficient vermifuge and, if properly pulverized, is harmless; and in some parts of Russia there is a popular belief that it is a valuable

[1] See page 300 in section on General Principles of Toxicology.
[2] *Virchow's Jahresbericht*, 1893, vol. i., p. 505.
[3] *Med. Record*, vol. lxiv., p. 25.
[4] *Lancet*, February 19, 1825, p. 217.

remedy in certain diseases. Death, however, has sometimes resulted from its use for this purpose.[1]

Observations made by one of the writers (Haines) in conjunction with Dr. E. F. Ingals on a professional glass-eater are of interest in this connection. He was a strong young negro who claimed and appeared to be in excellent health. He gained a livelihood by exhibiting himself in cheap museums and at fairs as a glass-eater. He voluntarily placed himself in our hands for scientific investigation. On one occasion he ate, in our presence and at our office, the following articles, all being provided by us personally: half a dozen six-inch test-tubes, two good-sized lamp chimneys, an ordinary four-ounce medicine bottle, two pieces of window-glass, each four inches square, and three slips of colored glass each an inch wide and three inches long. In eating them he bit the glass off, chewed it up, and swallowed it much as if it had been any ordinary article of food. He asserted that he could distinguish by the taste glass of different colors, and it was for this purpose that the colored slips were given him. He was thoroughly blindfolded when he ate them, but he promptly indicated the color.

Before and after eating the glass the man's mouth and throat were carefully examined by Dr. Ingals with a mirror and laryngoscope. They were found perfectly normal except that the gums and inside of the cheeks were whiter and thicker than normal, and, after eating the glass, they were bleeding from a number of small cuts. The man was kept under observation for several hours after eating the articles mentioned, but he at no time showed the least unfavorable symptom. At the end of the first hour slight emesis was provoked by tickling the fauces. The vomited matter consisted of partially digested food mixed with a large number of fragments of glass and an excessive amount of thick mucus. The man admitted that he had eaten a hearty meal before submitting to the test, and that this was his custom before each exhibition. He died two or three years subsequently from a subacute gastro-enteritis, presumably from the irritation produced by his long-continued glass-eating.

Gracy[2] reports another case of glass-eating very similar to the above, and the experiments of Lesauvage, published at Paris in 1810 in an inaugural dissertation,[3] are of similar import. He states that $2\frac{1}{2}$ drams of pounded glass were given to a cat without injury, and that a dog took 6 or 7 ounces in eight days without suffering the slightest inconvenience, although care was taken to administer it while the animal was fasting and the fragments were frequently a line in length. He himself swallowed a considerable number of pieces, up to 2 millimeters (0.08 inch) long, without producing any bad effect.

A consideration of such facts as the above has led some medicolegal authorities (compare the first case under Illustrative Cases, below) to question altogether the dangerous properties of pounded glass, forgetting the

[1] *Virchow's Jahresbericht, loc. cit.*

[2] *Med. and Surg. Reporter*, 1888, vol. lviii., p. 61.

[3] Lesauvage, *Recherches sur les effets du verre et des substances vitriformes portés à l'intérieur des organes digestifs.*

well-authenticated cases of serious sickness and death from the agent and overlooking the circumstance that, in the experiments of Lesauvage and of others in the same line, the largest pieces of glass administered were still quite small. After carefully examining the evidence concerning the subject we agree fully with the following conclusions reached by Marc[1] in regard to the question: "The observations which have been made on glass-eaters and the experiments of Caldani and Mandruzzato, which tend to demonstrate the harmlessness of substances of this kind, have been adopted too lightly by certain physicians. Caldani experimented on animals, and even, what appears difficult to conceive, on a young man of fifteen years, whom he caused to swallow powdered glass without his experiencing the least inconvenience. Mandruzzato repeated these same experiments on animals and on himself and obtained the same results. These observations, however, only prove at the most that powdered glass introduced into the stomach is not always harmful: and the above isolated facts do not demonstrate in any manner that in other cases and under other circumstances one or more sharp points applied to the interior walls of the alimentary canal may not produce a most serious mechanical action. It follows, moreover, from the fact that most glass-eaters die from intestinal affections[2] and from several sudden deaths following the swallowing of glass[3] that substances of this character may be dangerous."

The organism seems to be endowed with an instinctive provision by which it defends itself from threatened perforation by sharp articles. According to the observations of Exner,[4] when a sharp object approaches the wall of the intestine, the latter retreats from it, forming a sort of dimple which receives the sharp projection and prevents injury to the mucous membrane. Foreign bodies of this character are arranged by the action of the muscular walls of the intestine so that they lie with their longest axis parallel to the long axis of the section of the bowel in which they are situated, and thus the chance of injury is reduced to a minimum.

Mode of Action.—Sharp-cornered articles may act in several ways: First, the piece may cause perforation of the wall of the stomach or bowel, with the usual consequence of such perforation. Owing, however, to the defensive actions on the part of the intestine, spoken of above, perforation by such articles is relatively rare. The effects of perforation vary according to the size, shape, and character of the perforating body, as well as with the location in which the perforation occurs and the condition of the patient as to resisting power, etc. It is not uncommon to have needles penetrate the wall of the bowel without any noticeable results. If a perforation can be shown to have caused death, the presence of glass in the stomach or intestines or in the peritoneal cavity would raise a strong presumption in favor of the glass being the cause of death.

[1] *Manuel d'autopsie cadav. médico-lég.*, p. 61. [2] Plouequet, *Sur les morts violentes.*
[3] Gmelin, *Dans son histoire des poisons minéreaux*, and Metzger.
[4] *Arch. f. d. ges. Physiol.*, vol. lxxxix., p. 258.

Second, the action of a large number of pieces may set up a widespread irritation and inflammation, arising from the subsequent local infection, causing an acute or subacute gastro-enteritis, and this is probably the chief cause of suffering and death produced by these articles. It seems likely that in the case of professional glass-eaters the repeated wounding of the mucous membrane may have led to some sort of immunity which prevents an infection that might easily occur in other persons. The impunity with which these persons swallow such articles can hardly be urged against the possibility of death being produced in other persons. Especially the enfeebled condition of age or the presence of disease may render infection of slight wounds fatal. Before sickness or death, however, is attributed to the irritation caused by powdered glass, it should be shown that the other more usual causes of gastro-intestinal inflammation were absent and that the glass was administered in sufficient quantity and in particles of sufficient size to produce great irritation.

Third, the wound produced by the piece may become infected, and the infection spreading to other organs or involving the general system, may be fatal, and this is probably the cause of trouble in some cases. If evidence can be produced to show that the death arose from a remote infection proceeding from a wound of the gastro-intestinal mucous membrane, the presence of glass in the stomach or intestines would make it probable that it was the noxious agent. Here, also, immunity, original or acquired, may play a part.

Symptoms.—The symptoms produced by swallowing broken glass are generally due to irritation of the mucous membrane of the stomach and intestines, and are consequently similar to those of gastro-enteritis. They may vary in intensity from those cases in which but little inconvenience is felt to those attended with the most intense suffering. There is generally sharp pain in the stomach and later in the intestines, and sometimes nausea and vomiting, the vomited material usually being streaked with blood. The bowels are generally constipated, but sometimes there is diarrhea, the passage of stools being attended by pain, and the material passed is usually mixed with blood. In case of perforation of the stomach or intestines collapse usually supervenes and the patient soon dies.

In a case reported by Blake[1] a girl of sixteen, with suicidal intent, took a teaspoonful of broken glass mixed with bread. The next morning sharp cutting pains began to be felt, which were paroxysmal in character and referred to the epigastrium and later to the neighborhood of the umbilicus. When seen by the physician she was delirious from the pain, which required a hypodermic of morphin for its relief. There was great tenderness over the abdomen, which was somewhat swollen. The teeth chattered, the skin was hot and dry, the mouth and lips were parched, and the tongue clean, but very dry. There was excessive thirst; the pulse was 112 a minute and the temperature 99° F. The pain and tenderness continued for four days, when she had a copious stool following an injection of oil. The dejection was accompanied

[1] *Boston Med. and Surg. Jour.*, 1871, vol. lxxxiv., p. 191.

by pain and the passage of blood. The stool contained about a dozen large fragments of glass and a larger number of smaller pieces. From this time there was gradual improvement, and the patient was discharged from the hospital on the thirteenth day.

If the patient survives the immediate effects of the irritant, a subacute or chronic gastro-enteritis may follow which may last for weeks or months.

Fatal Dose and Fatal Period.—It is impossible to state what the fatal dose of broken glass is. As has already been pointed out, persons may take large quantities of the substance without any seemingly harmful results. On the other hand, smaller quantities may possibly produce great irritation and possibly perforation and death. Very much depends on the condition of the stomach, as to the presence or absence of food, upon the size and sharpness of fragments of glass, and on individual susceptibility. There is no doubt that a teaspoonful of pounded glass would in most cases be capable of producing serious effects, especially if the particles were of considerable size and presented numerous sharp angles; but the same quantity if finely pulverized might usually be taken with little or no effect. In a case reported by Hebb[1] a large teaspoonful of pounded glass caused the death of a child of eleven months.

Nothing can be said definitely in regard to the fatal period. The patient may die from perforation of the stomach or bowels or from acute gastro-enteritis in a day or two, or he may linger with subacute or chronic trouble for many days or weeks, and finally die from the remote effects of the irritant. In a fatal case reported by Reichardt[2] death occurred six days after taking the powdered glass.

Treatment.—If the case is seen early, an effort should be made to evacuate the stomach. This, however, cannot generally be accomplished by the stomach-tube or pump on account of the choking of the tube by the particles of glass. Resort must be had, therefore, to emesis, which is best produced by tickling the fauces or by hypodermic injection of apomorphin. There is some objection, however, to this treatment, for, in the act of vomiting, particles of glass may cut the walls of the stomach and of the esophagus; we believe, however, that, on the whole, it is better to evacuate the stomach even though there is some risk attending it. After evacuating the stomach, considerable quantities of bread, potatoes, or other similar food should be given to form a soft envelop for the pointed particles. If the bowels do not move, an injection of a mucilaginous or oily material should be used.

Pain should be treated by the use of opiates and hot applications, and collapse guarded against by the external use of warmth and the internal administration of cardiac and general stimulants, such as small doses of alcohol, ether, ammonium carbonate, and strychnin.

If the symptoms point to perforation of the stomach or bowels, a surgical operation should be promptly resorted to, with the hope of averting what would otherwise almost certainly be fatal.

[1] *Midland Med. and Surg. Reporter*, 1829, vol. i., p. 47.
[2] *Arch. d. Pharm.*, second series, vol. xci, p. 9.

Postmortem Appearances.—These are such as are commonly observed in cases of gastro-enteritis and are found almost entirely in the gastro-intestinal tract. There is redness of the mucous membrane, which is covered with tenacious mucus and frequently streaked with blood. Perforations in the stomach and intestines should be carefully looked for, and in case they are found, the abdominal cavity should be searched for particles of glass which may have escaped into it.

Hebb[1] gives the following account of the postmortem appearances in the case of an infant destroyed by the administration of roughly pounded glass: All the organs, including the large and small intestines, were normal except the stomach, which "was lined with a thick layer of tenacious mucus, streaked with blood, and it required to be peeled off before the villous coat beneath could be exposed to view. This last was in a state of amazing vascularity; it presented one entire congeries of vessels; numberless particles of glass, some large and some small, were adhering to it, and in many places it was lacerated by them."

In Reichardt's case[2] erosions were found in the pharynx, stomach, duodenum, and upper part of small intestines; and in the case of a boy of twelve years, described by Bronowski,[3] many ulcers were found on the mucous membrane of the stomach and intestines, and in both organs the powdered glass was discovered in a form resembling sand.

In making the postmortem examination unusual care should be exercised to determine the existence of wounds and to ascertain the situation of any pieces of glass that may be present in the stomach or intestines. In case of a legal investigation, much may depend upon these points in determining whether death occurred from the glass or possibly from other causes.

Chemical Examination.—The contents of the stomach and intestines and vomited matter and feces may be washed in a stool-sieve and any particles left on the sieve examined as directed hereafter. If the amount of food, however, is great or the amount of glass small, it is better to destroy the organic matter by treatment with hydrochloric acid and potassium chlorate by the process of Fresenius and Babo, as described on page 325 in the section on General Principles of Toxicology. After the destruction of the organic matter the material should be strained and particles of glass looked for in the residue. If metallic articles, such as tacks, needles, and pins, are to be searched for, the acid and chlorate are inadmissible and should be replaced by a strong solution of potassium hydroxid.

Particles of glass found by any of these methods should be carefully measured. They may be identified as glass by the following properties and tests: (*a*) They are transparent or translucent; if the glass, however, has an exceedingly irregular surface, this property may be largely lacking. (*b*) The surface is more or less irregular, with sharp, uneven angles, by which it may be distinguished from regularly crystalline bodies. (*c*) It is fusible when heated on platinum foil; sand or other forms of silica

[1] *Loc. cit.* [2] *Loc. cit.* [3] *Virchow's Jahresbericht*, 1893, vol. i., p. 505.

do not fuse. The globules adhere to the foil and may show sufficient transparency for lines to be seen through them and even for the reading of letters. (*d*) It is ductile. (*e*) When glass is heated in a platinum dish with hydrofluoric acid, it dissolves and the silica disappears, leaving a residue consisting of the fluorids of the metals from which the glass was made. Quartz or silica, for which glass might be mistaken, would disappear wholly under this test. The examination of this residue for metals may show the original composition of the glass.

In the examination the greatest care must be taken that the material shall not be contaminated by pieces of glass accidentally derived from vessels or other apparatus. The discovery of a few pieces of glass in the stomach may sometimes be due to the accidental swallowing of pieces of glass from glass vessels in which food has been contained.

Illustrative Cases.

Beck[1] reports a case that occurred near Bayeux, France, in which the accused was supposed to have murdered his wife with pounded glass. A quantity of this substance was found at the autopsy in the stomach and intestines, and these organs presented signs of marked irritation. Professors Baudelocque and Chaussier, who were called in to give testimony in the case, gave it as their opinion, after thoroughly considering all that had been written on the subject, that pounded glass is not dangerous, and suggested that the glass found in the stomach might have come from some vessel of that material being broken by the deceased's teeth during the convulsions which preceded her death. The accused was acquitted.

Dr. W. Turner, of Spanishtown, Jamaica, relates that an attempt was made by a negro woman to "poison" a family of seven with pounded glass. A large quantity of the pounded glass was put into a dish of curried fowl, of which every one ate freely. The presence of the glass being discovered toward the conclusion of the meal, the head of the family immediately administered purgatives. In consequence they all passed large quantities of coarsely powdered bottle-glass. When Dr. Turner saw them four days after the attempt, none of them had suffered any inconvenience.[2]

Portal[3] records a case in which a young man broke his drinking-glass with his teeth and swallowed the pieces. Frightful pains in the stomach and convulsive movements followed. Portal, who was called in as a physician, ordered the patient to eat much boiled cabbage to envelop the glass and then take an emetic, whereat many pieces of glass were thrown up. The patient then received milk, warm baths, and enemata, but in spite of this became emaciated and had to be kept on a milk diet for four weeks before he recovered.

Foderé,[4] arguing for the dangerous properties of pounded glass, men-

[1] *Medical Jurisprudence*, third edition, p. 507.
[2] *Edinburgh Med. and Surg. Jour.*, 1824, vol. xxii., p. 225.
[3] *Arch. d. Pharm.*, second series, vol. xci., p. 9.
[4] *Traité de médicine légale*, vol. iv., p. 113.

tions a case of a man who introduced a liqueur glass into his rectum. A surgeon, in endeavoring to remove it, broke it and was unable to extract one of the fragments. The latter caused intense pain, and produced several fistulous abscesses and extensive gangrene, and finally death. The author concludes with the question: "Would the evil results have been less if a piece of glass had remained in the stomach or any other part of the alimentary canal?"

At the trial of Mrs. Mary B. Sanderson, accused of killing her husband by feeding him ground glass in oatmeal, held in Marshall, Mich., December 19, 1899, F. F. Stevenson testified to having himself eaten ground glass without harm or inconvenience and also to having administered it to dogs without causing any injury. He exhibited pieces of glass from one-fourth to one-half inch in length which had passed through the alimentary canal of a dog without inconvenience to the animal. Dr. John E. Clark, Professor of Chemistry in the Detroit Medical College, gave similar testimony. Professor A. B. Prescott, who made the examination of the organs for the State, found five pieces which he recognized as glass by their transparency, their fusibility, their angular outline before fusion, and their globular character after fusion. The largest of these pieces was a little more than 2 mm. (0.08 in.) in diameter, the others ranging from ½ to 1½ mm. The deceased, who was eighty years of age, had been suffering for several weeks with diarrhea, the passages being tinged with blood. The accused was acquitted.

Marcet[1] reports the case of a sailor who was accustomed to swallow knives and other sharp objects; upon postmortem examination some thirty knife-blades were found in his stomach, but no evidence of recent or old ulcers.

Credé collected 26 cases in which the stomach had been opened for the extraction of foreign bodies, and Fricker[2] added 27 more. The latter reports a remarkable case of a woman who, in several attempts at suicide, swallowed numerous foreign bodies. He removed from her stomach 37 articles, including 1 key, 2 teaspoons, 1 fork, wire nails, 2 hairpins, 9 sewing needles, 12 pieces of glass, etc. After the removal of these bodies the patient made a rapid recovery. Some of these articles had remained in the stomach for three months.

Meissenbach[3] extracted a large number of articles, including an ounce of broken glass (electric-light globe), from the stomach of a professional glass-eater who styled himself "The Human Ostrich." Although he had swallowed glass and other foreign objects for a number of years, he had experienced no inconvenience until shortly before the operation was performed.

W. S. Halsted[4] reports a case in which he extracted from the stomach of a professional glass-eater, who had been induced by some

[1] *Medico-Chir. Trans.*, vol. xii., p. 72.
[2] *Deutsch. med. Wochenschr.*, 1897, No. 4, p. 56.
[3] *Jour. Amer. Med. Assoc.*, March 5, 1898, p. 513.
[4] *Contributions to the Science of Medicine*, p. 1047; the Johns Hopkins Press, 1900.

PLATE 15.

Seventy-four grams of glass extracted from the stomach (Halsted).

medical students to swallow a large number of foreign articles, 74 grams (1142 grains) of broken glass (Plate 15), and 208 articles consisting of 28 pieces of chain, 99 nails, and 81 screws, knives, tacks, pins, etc.

Another case similar to the above has been reported by G. F. Inch.[1]

Ollivier records[2] the case of an infant two and one-half months old who was given some pins by his nurse, who was accused of trying to murder him. The child passed the pins *per anum* and his health was not seriously affected. The accused stated that she acted under morbid impulse due to menstruation, and she was acquitted. Ollivier concludes that although pins or needles when swallowed may cause death, they seldom do so. A case is cited by him, however, in which an infant died from the effect of a needle which perforated the wall of the stomach.

[1] *Amer. Med.*, April 12, 1902, p. 603. [2] *Ann. d'hygiène*, 1839, vol. xxi., p. 178.

THE RESPONSIBILITIES OF PHARMACISTS AND THEIR AGENTS.

DRUGS and medicines are in all countries admitted to be necessary. They are valuable when properly used, but liable to prove injurious, dangerous, or even fatal to health and life when abused or ignorantly used, or when one medicinal substance is substituted for another through ignorance, accident, or design.

The public is necessarily held to be powerless to protect itself against the dangers attending the preparation, vending, and use of drugs, since special training, knowledge, and experience such as the public cannot possess are necessary to guard against those dangers.

"In the purchase of drugs the customer must rely upon the druggist to furnish the article called for; he is not held to the rule that he must examine for himself. In the very nature of things the druggist is held to warrant that the drugs or medicines he sells are of the character called for."[1]

For these reasons all civilized countries have, for the protection of the people, enacted special laws to govern and restrict the preparation and vending of drugs and medicines. These public health laws, and the regulations made thereunder, invariably provide that only persons specially licensed for that express purpose shall be permitted to dispense or sell medicines to the public, and that these specially licensed venders and dispensers of medicines shall exercise their rights and duties only under certain prescribed restrictions.

The legally licensed venders and preparers of medicines are commonly called registered pharmacists, and are also generally called druggists; but some State laws make specific distinction between "druggists" and "registered pharmacists," conferring upon the latter certain rights which are denied to "druggists." Such distinction is made in the State of New York, where licensed "pharmacists" are authorized to practise in cities of over 1,000,000 inhabitants, while licensed "druggists" are not permitted to practise pharmacy except in cities of less than 1,000,000 inhabitants.

Since the sole object of any law regulating and restricting the practice of pharmacy, or the preparation and dispensing or vending of drugs and medicines to the public, or directly or indirectly to persons by whom they are to be used, must be the proper protection of health and life, it is evident that the exclusive right conferred upon registered pharmacists is merely a part of the means by which the end in view may be attained,

[1] Jones *vs.* George, 56 Tex., 149.

and that the qualifications, duties, obligations, and responsibilities imposed upon them in return for that exclusive right constitute the only features of value to the people. All pharmacy laws, therefore, demand that the registered pharmacist shall be educated and trained in such manner that he is competent to perform his duties properly and safely, and that he shall, in fact, actually perform his duties with a degree of faithfulness and care proportionate to the danger involved.

"A state statute which requires the retailer of drugs, medicines, etc., to submit to an examination and procure a license is within the police power of the State and is not a tax on business, nor does it deprive one of property without due process of law."[1]

"The fee to be paid by the applicant for a license to engage in the business of an apothecary and druggist is merely an equivalent for the service rendered by the commissioners in making the examination and issuing the license, and cannot be considered as a tax upon the business, or as depriving the applicant of his property without due process of law."[2]

The educational qualifications of pharmacists and their assistants are in most countries defined by law. In the United States these qualifications are left wholly to the discretion of the Boards or Commissions of Pharmacy created by the pharmacy laws for the express purpose of securing to the people the protection sought. The powers conferred upon the Boards or Commissions of Pharmacy in that respect are, as a rule, ample, and wherever sufficiently supported by public opinion, may be exercised so as to establish whatever standard of education and efficiency they may deem necessary to the welfare of the community.

In many countries the pharmacists are not only obliged to conform to certain prescribed educational requirements, but are also required to make oath before they enter upon their duties that they will faithfully discharge their obligations without fear or favor, uninfluenced by pecuniary loss or gain or by other considerations. In those countries, also, the drug-stores or pharmacies, as well as the pharmacists, are registered and annually or oftener inspected. New York is the only State in America where pharmacies or drug-stores are required to be registered.

The governments of several countries require every pharmacy to be at all times prepared to furnish, without unnecessary delay, specified medicines declared by properly constituted medical authority to be especially important, so that physicians know what remedies are everywhere available in emergencies.

The sale of poisons is regulated by law in all countries. In America the poison laws differ essentially in different States. Sometimes they contain enumerations of the particular substances classed as poisons; but such enumerations are of little value, because other substances generally recognized as poisonous are subject to the same restrictions whether mentioned by name or not. The poisons usually named are such substances as have been known to be used for suicidal and other

[1] State vs. Heinemann, 80 Wis., 258.
[2] State vs. Forcier, 65 N. H., 42.

criminal purposes, and substances such as opium, morphin, cocain, chloral, and others, the use of which may lead to habitual abuse. As nearly all poisons are valuable medicines when properly used, and nearly all the most valuable medicines are poisons when abused or ignorantly used, only pharmacists are permitted to sell poisons, and even the pharmacists are forbidden to sell poisons except when ordered for medicinal purposes by physicians, or under restrictions and precautions specified in the law.

"In a case where a druggist who, at the request of a customer, dropped croton oil on a piece of candy which the purchaser then gave to another person who ate the candy so drugged to his serious inconvenience and injury, the druggist knowing or having reason to believe that the dose was intended for such person or some one else, not for medicinal purposes, but 'as a drug,' that druggist was held guilty of assault and battery."[1]

Standards of identity, purity, and strength of all important and commonly used medicines are established by national pharmacopeias in almost all civilized countries. Deviations from these standards are violations of law which, if injury results therefrom, render persons responsible for them liable to punishment or damages. In some States such offenses are specially covered by laws against the adulteration of food and drugs. In States where no specific laws exist, the pharmacist is, nevertheless, liable for any injury or damage which may result from the sale, by him, of any drug or medicine which may not be that properly implied by the name under which it is sold, whether the substitution be due to ignorance, accident, or design.

"It is the duty of the druggist to know the properties of his drugs and to be able to distinguish them from each other. It is his duty so to qualify himself, or to employ those that are so qualified, to attend to the business of compounding and vending medicines and drugs, that one drug may not be sold for another. He cannot escape responsibility on the pretext that the use of one drug for another was an accidental or innocent mistake. It will not avail him to claim that he had used extraordinary care and diligence in preparing or compounding the medicines as required, etc. He is liable at the suit of the party injured, for damages at the discretion of a jury."[2]

The education and training of the pharmacist, by virtue of which he is registered as such and given the exclusive right to practise his occupation, are not exacted without the implied duty on his part to exercise them for the benefit of the people. He is, therefore, properly held responsible for the identity and proper character and condition of the medicines he furnishes, for if the licensed pharmacist is not required to render any service or perform any duty which entitles him to enjoy the exclusive right to practise his occupation, but is only an irresponsible agent, then the special object of the pharmacy law is wholly defeated. He must know of his own knowledge what he is dispensing and how it should be dispensed, and must do his work with due care. He must

[1] State *vs.* Munroe, 121 N. C., 677. [2] Fleet *vs.* Hollenkemp, 52 Ky., 219.

verify the identity, quality, purity, and strength of all medicinal substances dispensed or sold by him so far as these admit of such verification by the means expressly prescribed for that purpose by recognized authorities. The Pharmacopeia is the authority concerning all medicinal substances enumerated in it, and is so recognized by the courts as well as by the executive branches of the Government of the United States and by the various branches of the State governments.

"Druggists are liable in damages to the party injured for negligence or want of skill in the compounding or sale of medicines. Druggists, apothecaries, and all persons engaged in manufacturing, compounding, or vending drugs and medicines should not only be required to be skilful, but should also be exceedingly cautious and prudent. All persons who deal with deadly poison and noxious and dangerous substances are held to a strict accountability. A druggist is held to a special degree of responsibility. The care required must be commensurate with the danger involved, and the skill to be exercised must correspond with that superior knowledge which the law requires. If in compounding a medicine the druggist grinds the different articles of which it is composed in a mill used to grind poisonous drugs without properly cleansing the mill, he is liable for the injury caused thereby."[1]

If the pharmacist sells or dispenses morphin or strychnin when quinin is called for, he cannot escape responsibility for consequent disaster on the plea that he had received it labeled quinin, that he believed it to be just what the label indicated, and had no reason to believe otherwise. Courts have decided that it is his duty to subject the medicines he dispenses to proper tests and to detect mistakes and frauds.

"A dealer in drugs and medicines who carelessly labels a deadly poison as a harmless medicine and sends it so labeled into the market is liable to any person who, without fault on his part, is injured thereby, though it may have passed through other hands by intermediate sales before it reaches the person injured. The druggist owes to the public a duty not to expose human life to danger by falsely labeling a noxious drug and selling it as a harmless article. An imperative social duty requires of him that he use such precautions as may prevent death or injury to those who may, in the ordinary course of events, be exposed to the dangers incident to the traffic in poisonous drugs."[2]

"A dealer in drugs and medicines who carelessly labels a deadly poison as a harmless medicine and sends it so labeled into the market is liable to all persons who, without fault on their part, are injured by using it as such medicine in consequence of the false label. The liability of the dealer in such case arises not out of any contract or direct privity between him and the person injured, but out of the duty which the law imposes upon him to avoid acts in their nature dangerous to the lives of others. Where such act is done by an agent, the principal is liable for the injury caused thereby."[3]

[1] Fleet *vs.* Hollenkemp, 52 Ky., 219. [2] Thomas *vs.* Winchester, 6 N. Y., 396.
[3] Davis *vs.* Guarnieri, 45 Ohio St., 470.

"The label of a harmless drug placed by a reputable wholesale druggist on a poisonous drug will not protect from liability a retail druggist who fails to discover the mistake in handling that drug and negligently selling it as the harmless drug indicated by the label. No doubt an action would lie against the wholesale druggist who made the first mistake, but if the retail druggist breaks the original package, it is his duty to know and determine the character of the contents, and, if he is guilty of negligence in this respect, he must respond."[1]

"If a druggist negligently sells a deadly poison as and for a harmless medicine to a customer who buys it to administer it to another person and who gives such other person a dose of it as a medicine from which such person dies, a right of action in tort arises against the druggist to the estate of the deceased."[2]

If the attending physician in a critical case decides that the only means by which he might be able to save the patient's life is the immediate administration of as large a dose as may be given of an extremely potent well-known remedy, then the patient's life may be lost if the pharmacist dispenses an article of twice the prescribed strength or one which has become wholly inert. Such results may easily follow when hydrocyanic acid is dispensed which has not been properly tested.

Another extremely valuable and potent remedy sometimes prescribed in heroic doses is extract of physostigma. According to some pharmacopeias, it is prepared by extracting the drug with diluted alcohol; other pharmacopeias prescribe undiluted alcohol. The extract made with strong alcohol is about four times as strong as the other. If the physician orders this extract, basing the dose upon results obtained with the weaker preparation, and the stronger extract is dispensed, a human life may be lost instead of saved; and if, on the other hand, he has become accustomed to the effects of the stronger preparation and prescribes one-fourth as much as would be required of the weaker extract, the patient's life might easily be lost for want of a sufficient amount of the only remedy available should the weaker extract be dispensed. The pharmacist must, in such a case, know what extract is the proper one, and must also know enough not to dispense a fatal dose. If the extract he dispenses was not prepared by himself, he must test it, which any competent pharmacist can readily do.

There are many pharmaceutic preparations of such a nature that they cannot be reliably tested, but a great proportion of the medicines ordered by physicians are such that their quality and potency can be determined by well-known scientific methods familiar to all really competent pharmacists, and those medicines, fortunately, include nearly all the most valuable and potent remedies known.

Whenever any physician orders, or any person procures, from the pharmacist any patented medicine, or one made by a patented process, or protected by a copyright, or a proprietary preparation, or a preparation made by a private formula, or a medicine whose identity or composition is in any way kept secret, so that full and free examination of

[1] Howes *vs.* Rose, 13 Ind. App., 674. [2] Norton *vs.* Sewall, 106 Mass., 143.

its nature and properties is prevented, or whenever the prescriber or buyer directly or indirectly specifies the product of any particular manufacturer, then the pharmacist who furnishes the article is relieved of all responsibility for it *in his capacity as a pharmacist*, because his functions as such are in no way concerned in the mere sale of an article whose quality, purity, composition, potency, age, or condition he cannot control or verify. All that the pharmacist can do in such a case is what any merchant can do just as well—to furnish precisely what is ordered, using reasonable care not to supply an article that is damaged.

"In the sale of patent or proprietary medicines furnished by the compounder of the ingredients which compose them, the druggist is not required to analyze the contents of each bottle or package he receives. If he delivers to the consumer the article called for with the label of the proprietor or patentee upon it, he cannot be justly charged with negligence in regard to it."[1]

The most important service which the pharmacist is alone competent and permitted to render is that of preparing or dispensing medicines ordered by physicians. Physicians are not required to possess that special pharmaceutic knowledge, skill, and experience which are necessary, and are, therefore, as a rule, not permitted to practise pharmacy or, in other words, to prepare and dispense medicines. In States where the law does not enjoin upon the druggist a special pharmaceutic training such as tends to invest his peculiar technical services with greater value as a safeguard against danger, the restriction of the practice of pharmacy to his hands would be without warrant.

A physician's prescription is simply his order to any authorized pharmacist to prepare and dispense the remedy or remedies prescribed in whatever manner directed. It is, therefore, not the property of the patient. The patient compensates the physician for diagnosis, advice, and treatment; but any prescription which the physician may write for medicines is simply a detail incident to the treatment. The patient does not buy, and the physician does not sell, prescriptions. Whenever the prescription has been presented to a duly licensed pharmacist and the directions carried out, that pharmacist may properly retain the prescription in his possession on the ground that it is an order to him. It follows from this view of the matter that the pharmacist has no right to dispense the medicine more than once unless the writer of the prescription expressly so directs, and he has no right to dispense that medicine at all if to his knowledge it is intended to be used for some person other than the one for whom it was prescribed, or for some purpose other than that for which it was intended by the prescriber, nor has he any right to dispense a prescription for any potent remedy when presented so long after the date upon which it was written that reasonable doubt may exist as to whether the physician would again prescribe the same medicine after the lapse of time between the date of the order and the date on which it is presented to the pharmacist.

Prescriptions are written not only by competent physicians, but also

[1] West *vs.* Emanuel, 198 Pa. St., 180.

by incompetent ones and by persons who are not legally licensed practitioners of medicine. Errors may be committed by any of these writers of prescriptions, and these errors are often of such a nature as to lead to serious or even fatal consequences should the pharmacist dispense the medicines as ordered. In cases where the errors occurring in prescriptions must be regarded as self-evident to a competent and careful pharmacist, the dispenser is rightly expected to detect and avoid them, and if himself negligent, is not to be relieved of responsibility by the error, negligence, or ignorance of the prescriber.

The dangerous defects in prescriptions may be classified as follows: (1) Writing the names of other medicines in place of those actually intended; (2) dangerous abbreviations of the names employed; (3) accidental transpositions of parts of prescriptions or of the directions given concerning the uses of the medicines ordered; (4) chemical and other incompatibilities between any two or more of the medicinal substances ordered to be combined with each other; and (5) dangerous doses.

The following examples may suffice to illustrate the several classes of defects mentioned:

1. Extracts are often many times as potent as fluid extracts. Therefore, if a prescription calls for the extract instead of the fluid extract, the word "fluid" being inadvertently omitted, it is the duty of the pharmacist to detect the omission if the dose prescribed of the extract is clearly a dangerous one.

2. Dangerous abbreviations of the titles of medicines are not uncommon. Thus the abbreviation "Hydr. Chlor." may mean Hydrargyri Chloridum Mite (calomel), or it may mean Hydrargyri Chloridum Corrosivum (corrosive sublimate), or it may mean Hydras Chloralis (hydrate of chloral). These are widely different substances with different medicinal properties and doses. The abbreviation "Aquæ Acidi Carb." may mean either carbonic-acid water or carbolic-acid water, the latter quite dangerous if used in place of the former. The abbreviation "Elat." may stand for elaterium or for the several times more active elaterin, and both substances are so potent as to be dangerous if used in large doses.

3. Transpositions of the quantities ordered of different ingredients of any mixture are not uncommon, because many physicians naturally write the names of the remedies first and afterward decide what quantities they will order, and in writing these quantities after the several ingredients they may accidentally transpose the doses of dangerous and relatively harmless substances.

4. Physical and chemical incompatibilities between the ingredients of a mixture are commonly occurring defects in prescriptions. If the ingredients are of such character that they act upon each other chemically, so that new substances are formed which the prescriber did not expect would be formed, the pharmacist as well as the prescriber is responsible for any injury resulting should the chemical change be one that may fairly be regarded as readily foreseen or detected by any care-

ful person possessing the chemical knowledge indispensable to pharmacists.

An example of dangerous chemical and physical incompatibility in a prescription is furnished by the fatal result of dispensing strychnin sulphate and potassium bromid dissolved in water and then mixed together, which led to the separation of strychnin hydrobromid at the bottom of the bottle, so that nearly all the mixture was consumed before any strychnin was taken, after which all that poisonous substance was taken at once in the last dose, alkaloidal salts being generally insoluble in strong water solutions of inorganic salts like potassium bromid. The responsibility of the dispenser for any injury resulting from incompatibilities in prescriptions must be determined in every case according to its own merits, and he can be held for damages only in cases where it may be fairly shown that the injury was due to culpable ignorance or carelessness on his part.

5. Dangerous doses may be dispensed with serious results through the error or carelessness of prescriber or dispenser or both. The pharmacist is not only justified in refusing to dispense a dose which in his opinion is evidently likely to prove fatal, but it is his duty not to dispense it.

When a prescription is dispensed, the responsibility for the character of the medicinal substances used rests upon the licensed pharmacist in charge, while the responsibility for the skill and accuracy of the technical work involved in the preparation, compounding, or dispensing of the medicine rests both upon the person who actually renders that service and upon the employer. There are necessarily everywhere three distinct grades of pharmaceutic workers in the drug-stores: (1) Registered pharmacists in charge; (2) registered pharmacists and assistant pharmacists employed under the direction of the pharmacist in charge; and (3) apprentices.

According to the laws of most of the States of America the proprietor or proprietors of a drug-store may or may not be licensed, no person being prevented from owning one or more drug-stores in whole or in part. But no person who is not a registered licensed pharmacist can lawfully conduct, manage, or "keep open" a drug-store or pharmacy or practise pharmacy. If the owner is not himself licensed, he must employ and place in charge of the drug-store a legally registered pharmacist; but this does not relieve the owner of responsibility for the conduct of the pharmacy or of liability for any injury or damage which may result from violation of law, ignorance, incompetence, or carelessness on the part of his agents or employees, nor for any damage due to avoidable accident. The owner and the registered pharmacist in charge, whether he be the owner or not, may be held responsible for the identity, quality, and strength of the medicinal materials employed in preparing and dispensing the medicines furnished in and from their stores. The registered pharmacist in charge is permitted to occupy his position by virtue of the assumption that he possesses the knowledge, skill, and experience necessary to render him competent to perform every duty

properly expected of a pharmacist, including the ability to verify the character of the medicines and test their quality, purity, and strength, and any registered pharmacist who accepts the position of pharmacist in charge of a drug-store or pharmacy thereby agrees to perform these duties.

"A Pennsylvania statute (Act of June 16, 1891, P. L. 313) provides that no person shall engage in the business of retailing drugs who has not obtained a certificate of competency from the State pharmaceutical examining board and been duly registered as provided by the statute; but further provides that the widow or legal representative of a deceased registered pharmacist may continue in the business of such deceased registered pharmacist. It was held that this statute is unconstitutional, that it is not a valid exercise of the police power, but is a species of class legislation, *in that it permits certain unqualified persons to engage in the retail drug business* and excludes others."[1]

Assistants are not held responsible for the character of the materials supplied by their employers, and which are used by said assistants without personal knowledge concerning their genuineness, quality, or strength, but any person who dispenses or sells drugs or medicines known to him to be fraudulent or adulterated or to deviate from the recognized standards renders himself *particeps criminis*.

All persons who prepare and dispense medicines for the public, whether as principals or agents, are responsible before the law for their own acts in the exercise of their functions under their licenses as pharmacists. In addition the principals are responsible for the acts of their agents.

"Druggists are responsible and liable for any negligence or incompetence of their agents or clerks."[2]

Apprentices in pharmacy are persons who are permitted to participate in the technical work and daily routine of pharmaceutic practice and in the vending of drugs and medicines, under the immediate direction and supervision of qualified licensed pharmacists and assistant pharmacists, but not without such direction and supervision. This permission to apprentices to assist in preparing and vending medicines is necessary in order that they may acquire in due time, without danger to the public, that practical mastery of the duties of pharmacy which is requisite to develop them into intelligent, safe, and reliable pharmacists prepared to take the places of their precursors. The law assumes or specifically directs that the persons employed as apprentices in pharmacy possess sufficient general education and intelligence to be fit to undertake to learn chemistry, pharmacognosy, and pharmacy.

A druggist or pharmacist may legally refuse to furnish any medicine which in his opinion is liable to do injury.

"The mere refusal of a druggist to fill a prescription furnishes no occasion to hold him for damages. In many cases he may have the best reasons for declining to do so. He may perceive or have cause to suspect that the physician erred in his prescription."[3]

[1] Com. *vs.* Zacharias, 3 Pa. Super. Ct. Rp., 264.

[2] Fleet *vs.* Hollenkemp, 52 Ky., 219. [3] Tarleton *vs.* Lagarde, 46 La. Ann., 1368.

A pharmacist is not responsible for injuries resulting from the misuse of any drug sold by him unless it can be shown that the seller had reason to believe that it would not be properly or safely used or the sale was one expressly prohibited by law.

"When a person who has reached the age of discretion and who is apparently in possession of his mental faculties applies to a druggist for a certain drug, he represents to the druggist, at least by implication, that he knows its properties and uses and that he is a fit person to whom sale thereof may be made, and unless there is something connected with the transaction or previously known to the seller indicating that the would-be purchaser cannot safely be intrusted with that drug, a sale of the substance called for may be made without explaining its properties or the manner in which it may be safely used or handled, and, under such circumstances, the seller is not liable in damages for injuries to the purchaser resulting from the improper use or handling of the article, no matter how little knowledge the purchaser may in fact have had of its properties or of the manner in which it could not be safely used or handled. The vender's legal duty to the purchaser in such a case can go no further than to give him the identical substance he calls for." [1]

In any action brought against a druggist or pharmacist for damages, actual injury must be shown to be the result of the unlawful act, ignorance, incompetence, or neglect on the part of the defendant or his agent or agents.

"In order that there may be a recovery of damages it must appear not only that the druggist was guilty of negligence, but that the person injured was free from negligence contributing to the injury." [2]

"The authorities do not appear to have gone so far as to dispense with actual negligence as a necessary element in the liability when a mistake has occurred." [3]

[1] Gibson *vs.* Torbert, 115 Ia., 163.
[2] Hackett *vs.* Pratt, 52 Ill. App., 374.
[3] Brown *vs.* Marshall, 47 Mich., 576.

INDEX TO VOL. II.

GENERAL INDEX.

Stelwagon's Diseases of the Skin

A Treatise on Diseases of the Skin. For Advanced Students and Practitioners. By HENRY W. STELWAGON, M. D., PH. D., Clinical Professor of Dermatology, Jefferson Medical College and Woman's Medical College, Philadelphia; Dermatologist to the Howard and to the Philadelphia Hospitals. Handsome octavo of 1115 pages, with 220 text-cuts and 26 full-page colored lithographic and half-tone plates. Cloth, $6.00 net; Sheep or Half Morocco, $7.00 net.

COMBINING A THOROUGH TREATISE WITH A COMPLETE ATLAS
SECOND EDITION—FIRST EDITION EXHAUSTED IN SIX MONTHS

The exhaustion of the first edition of this work in a period of six months, and the many complimentary review notices have been exceedingly gratifying. Such a kind reception permits the inference that the predominant aim kept in view in its preparation, of giving the general physician a treatise written on plain and practical lines, with abundant helpful case-illustrations, has been successful. While the short time that has elapsed since its appearance makes a revision unnecessary, the author has taken advantage of this opportunity to correct some typographical errors, and to change a few expressions that seemed of somewhat ambiguous meaning.

PERSONAL AND PRESS OPINIONS

John T. Bowen, M.D.,
Assistant Professor of Dermatology, Harvard University Medical School, Boston.

"It gives me great pleasure to endorse Dr. Stelwagon's book. The clearness of description is a marked feature. It is also very carefully compiled. It is one of the best text-books yet published and a credit to American dermatology."

Grover W. Wende, M.D.,
Clinical Professor of Dermatology, University of Buffalo.

"I have recommended that the work be included in the catalogue of the University of Buffalo. I think you are to be congratulated on bringing out one of the very best works published in the English language."

Boston Medical and Surgical Journal

"We can cordially recommend Dr. Stelwagon's book to the profession as the best text-book on dermatology, for the advanced student and general practitioner, that has been brought strictly up to date. . . . The photographic illustrations are numerous, and many of them are of great excellence."

DeSchweinitz's Diseases of the Eye

Recently Issued—Fourth Edition, Enlarged and Reset

Diseases of the Eye: A HANDBOOK OF OPHTHALMIC PRACTICE. By G. E. DESCHWEINITZ, M. D., Professor of Ophthalmology in the University of Pennsylvania, Philadelphia, etc. Handsome octavo of 773 pages, 280 text-illustrations, and 6 chromo-lithographic plates. Cloth, $5.00 net; Sheep or Half Morocco, $6.00 net.

WITH 280 TEXT-ILLUSTRATIONS AND 6 COLORED PLATES

In this new edition the text has been thoroughly revised, and the entire work has been reset. Many new chapters have been added, such as Thomson's Lantern Test for Color-Blindness Hysteric Alopecia of the Eyelids; Metastatic Gonorrheal Conjunctivitis; Grill-like Keratitis (Haab); the so-called Holes in the Macula; Divergence-paralysis; Convergence-paralysis, and many others. A large number of therapeutic agents comparatively recently introduced, particularly the newer silver salts, are given in connection with the diseases in which they are indicated. The illustrative feature of the work has been greatly enhanced in value by the addition of many new cuts and six full-page chromo-lithographic plates, all most accurately portraying the pathologic conditions which they represent.

PERSONAL AND PRESS OPINIONS

Samuel Theobald, M.D.,

Clinical Professor of Ophthalmology, Johns Hopkins University, Baltimore.

"It is a work that I have held in high esteem, and is one of the two or three books upon the eye which I have been in the habit of recommending to my students in the Johns Hopkins Medical School."

Late William Pepper, M. D.,

Professor of Theory and Practice of Medicine and Clinical Medicine, University of Pennsylvania.

"A work that will meet the requirements not only of the specialist, but also of the general practitioner in a rare degree. I am satisfied that unusual success awaits it."

British Medical Journal

"A clearly written, comprehensive manual. One which we can commend to students as a reliable text-book, written with an evident knowledge of the wants of those entering upon the study of this special branch of medical science."

Barton and Wells' Medical Thesaurus

A NEW WORK—JUST ISSUED

A Thesaurus of Medical Words and Phrases. By WILFRED M. BARTON, A. M., Assistant to Professor of Materia Medica and Therapeutics, and Lecturer on Pharmacy, Georgetown University, Washington, D. C.; and WALTER A. WELLS, M. D., Demonstrator of Laryngology, Georgetown University, Washington, D. C. Handsome 12mo of 534 pages. Flexible leather, $2.50 net; with thumb index, $3.00 net.

THE ONLY MEDICAL THESAURUS EVER PUBLISHED

This work is the only Medical Thesaurus ever published. It aims to perform for medical literature the same services which Roget's work has done for literature in general; that is, instead of, as an ordinary dictionary does, supplying the meaning to given words, it reverses the process, and when the meaning or idea is in the mind, it endeavors to supply the fitting term or phrase to express that idea. To obviate constant reference to a lexicon to discover the meaning of terms, brief definitions are given before each word. As a dictionary is of service to those who need assistance in interpreting the expressed thought of others, the Thesaurus is intended to assist those who have to write or to speak to give proper expression to their own thoughts. In order to enhance the practical application of the book cross references from one caption to another have been introduced, and terms inserted under more than one caption when the nature of the term permitted. In the matter of synonyms of technical words the authors have performed for medical science a service never before attempted. Writers and speakers desiring to avoid unpleasant repetition of words will find this feature of the work of invaluable service. Indeed, this Thesaurus of medical terms and phrases will be found of inestimable value to all persons who are called upon to state or explain any subject in the technical language of medicine. To this class belong not only teachers in medical colleges and authors of medical books, but also every member of the profession who at some time may be required to deliver an address, state his experience before a medical society, contribute to the medical press, or give testimony before a court as an expert witness.

Brühl, Politzer, *and* Smith's Otology

Atlas and Epitome of Otology. By GUSTAV BRÜHL, M. D., of Berlin, with the collaboration of PROFESSOR DR. A. POLITZER, of Vienna. Edited, with additions, by S. MACCUEN SMITH, M. D., Clinical Professor of Otology, Jefferson Medical College, Philadelphia. With 244 colored figures on 39 lithographic plates, 99 text illustrations, and 292 pages of text. Cloth, $3.00 net. *In Saunders' Hand-Atlas Series.*

INCLUDING ANATOMY AND PHYSIOLOGY

The work is both didactic and clinical in its teaching. A special feature is the very complete exposition of the minute anatomy of the ear, a working knowledge of which is so essential to an intelligent conception of the science of otology. The association of Professor Politzer and the use of so many valuable specimens from his notably rich collection especially enhance the value of the treatise. The work contains everything of importance in the elementary study of otology.

Clarence J. Blake, M. D.,
Professor of Otology in Harvard University Medical School, Boston.

"The most complete work of its kind as yet published, and one commending itself to both the student and the teacher in the character and scope of its illustrations."

Grünwald *and* Grayson's Diseases of the Larynx

Atlas and Epitome of Diseases of the Larynx. By DR. L. GRÜNWALD, of Munich. Edited, with additions, by CHARLES P. GRAYSON, M. D., Physician-in-Charge, Throat and Nose Department, Hospital of the University of Pennsylvania. With 107 colored figures on 44 plates, 25 text-illustrations, and 103 pages of text. Cloth, $2.50 net. *In Saunders' Hand-Atlas Series.*

In this work the author has given special attention to the clinical portion, the sections on diagnosis and treatment being particularly full. The plates portray, with a remarkable fidelity to nature, pathologic conditions that it would require a number of years to duplicate in practice. A knowledge of the histology of the morbid processes being essential to a proper understanding of them, twelve plates, showing the most important elementary alterations, have been included.

British Medical Journal

"Excels everything we have hitherto seen in the way of colored illustrations of diseases of the larynx. . . . Not only valuable for the teaching of laryngology, it will prove of the greatest help to those who are perfecting themselves by private study."

Haab and DeSchweinitz's External Diseases *of the* Eye

Atlas and Epitome of External Diseases of the Eye. By Dr. O. Haab, of Zürich. Edited, with additions, by G. E. deSchweinitz, M. D., Professor of Ophthalmology, University of Pennsylvania. With 98 colored illustrations on 48 lithographic plates and 232 pages of text. Cloth, $3.00 net. *In Saunders' Hand-Atlas Series.*

SECOND REVISED EDITION — JUST ISSUED

Conditions attending diseases of the external eye, which are often so complicated, have probably never been more clearly and comprehensively expounded than in the forelying work, in which the pictorial most happily supplements the verbal description. The price of the book is remarkably low.

The Medical Record, New York

"The work is excellently suited to the student of ophthalmology and to the practising physician. It cannot fail to attain a well-deserved popularity."

Haab and DeSchweinitz's Ophthalmoscopy

Atlas and Epitome of Ophthalmoscopy and Ophthalmoscopic Diagnosis. By Dr. O. Haab, of Zürich. *From the Third Revised and Enlarged German Edition.* Edited, with additions, by G. E. deSchweinitz, M. D., Professor of Ophthalmology, University of Pennsylvania. With 152 colored lithographic illustrations and 85 pages of text. Cloth, $3.00 net. *In Saunders' Hand-Atlas Series.*

The great value of Prof. Haab's Atlas of Ophthalmoscopy and Ophthalmoscopic Diagnosis has been fully established and entirely justified an English translation. Not only is the student made acquainted with carefully prepared ophthalmoscopic drawings done into well-executed lithographs of the most important fundus changes, but, in many instances, plates of the microscopic lesions are added. The whole furnishes a manual of the greatest possible service.

The Lancet, London

"We recommend it as a work that should be in the ophthalmic wards or in the library of every hospital into which ophthalmic cases are received."

American Text-Book of Genito-Urinary, Syphilis, Skin

American Text-book of Genito-Urinary Diseases, Syphilis, and Diseases of the Skin. Edited by L. BOLTON BANGS, M. D., late Prof. of Genito-Urinary Surgery, University and Bellevue Hospital Medical College, New York; and W. A. HARDAWAY, M. D., Professor of Diseases of the Skin, Missouri Medical College. Imperial octavo, 1229 pages, with 300 engravings, 20 colored plates. Cloth, $7.00 net; Sheep or Half Morocco, $8.00 net.

CONTAINING 20 COLORED PLATES

This work is intended for both the student and practitioner, giving, as it does, a comprehensive and detailed presentation of the subjects discussed. The work is original and fully representative. The illustrations, many of which are in colors, portray the conditions with rare fidelity, and will be found invaluable as an aid in diagnosis.

Journal of the American Medical Association

"This voluminous work is thoroughly up-to-date, and the chapters on genito-urinary diseases are especially valuable. The illustrations are fine and are mostly original. The section on dermatology is concise and in every way admirable."

Senn's Genito-Urinary Tuberculosis

Tuberculosis of the Genito-Urinary Organs, Male and Female. By N. SENN, M. D., PH. D., LL.D., Professor of Surgery in Rush Medical College; Attending Surgeon to the Presbyterian Hospital, Chicago. Octavo volume of 317 pages, illustrated. Cloth, $3.00 net.

MALE AND FEMALE

Tuberculosis of the male and female genito-urinary organs is such a frequent, distressing, and fatal affection that a special treatise on the subject appears to fill a gap in medical literature. In the present work the bacteriology of the subject has received due attention, the modern resources employed in the differential diagnosis between tubercular and other inflammatory affections are fully described, and the medical and surgical therapeutics are discussed in detail.

British Medical Journal

"The book will well repay perusal. It is the final word, as our knowledge stands, upon the diseases of which it treats, and will add to the reputation of its distinguished author."

Mracek *and* Stelwagon's Diseases of the Skin

Atlas and Epitome of Diseases of the Skin. By PROF. DR. FRANZ MRACEK, of Vienna. Edited, with additions, by HENRY W. STELWAGON, M.D., Clinical Professor of Dermatology, Jefferson Medical College, Philadelphia. With 63 colored plates, 39 half-tone illustrations, and 200 pages of text. Cloth, $3.50 net. *In Saunders' Hand-Atlas Series.*

CONTAINING 63 COLORED PLATES

This volume, the outcome of years of scientific and artistic work, contains, together with colored plates of unusual beauty, numerous illustrations in black, and a text comprehending the entire field of dermatology. The illustrations are all original and prepared from actual cases in Mracek's clinic, and the execution of the plates is superior to that of any, even the most expensive, dermatologic atlas hitherto published.

American Journal of the Medical Sciences

"The advantages which we see in this book and which recommend it to our minds are: First, its handiness; secondly, the plates, which are excellent as regards drawing, color, and the diagnostic points which they bring out."

Mracek *and* Bangs' Syphilis and Venereal

Atlas and Epitome of Syphilis and the Venereal Diseases. By PROF. DR. FRANZ MRACEK, of Vienna. Edited, with additions, by L. BOLTON BANGS, M. D., late Prof. of Genito-Urinary Surgery, University and Bellevue Hospital Medical College, New York. With 71 colored plates and 122 pages of text. Cloth, $3.50 net. *In Saunders' Hand-Atlas Series.*

CONTAINING 71 COLORED PLATES

According to the unanimous opinion of numerous authorities, to whom the original illustrations of this book were presented, they surpass in beauty anything of the kind that has been produced in this field, not only in Germany, but throughout the literature of the world.

Robert L. Dickinson, M. D.,

Art Editor of "The American Text-Book of Obstetrics."

"The book that appeals instantly to me for the strikingly successful, valuable, and graphic character of its illustrations is the 'Atlas of Syphilis and the Venereal Diseases.' I know of nothing in this country that can compare with it."

Grant's
Face, Mouth, and Jaws

A Text-Book of the Surgical Principles and Surgical Diseases of the Face, Mouth, and Jaws. For Dental Students. By H. Horace Grant, A. M., M. D., Professor of Surgery and of Clinical Surgery, Hospital College of Medicine; Professor of Oral Surgery, Louisville College of Dentistry, Louisville. Octavo volume of 231 pages, with 68 illustrations. Cloth, $2.50 net.

FOR DENTAL STUDENTS

This text-book, designed for the student of dentistry, succinctly explains the principles of dental surgery applicable to all operative procedures, and also discusses such surgical lesions as are likely to require diagnosis and perhaps treatment by the dentist. The arrangement and subject-matter cover the needs of the dental student without encumbering him with any details foreign to the course of instruction usually followed in dental colleges at the present time. The work includes, moreover, such emergency procedures as not alone the dentist and physician, but also the layman, may be called upon to perform. These, like the other subjects in the book, have been described in clear, concise language.

Grünwald *and* Newcomb's
Mouth, Pharynx, and Nose

Atlas and Epitome of Diseases of the Mouth, Pharynx, and Nose. By Dr. L. Grünwald, of Munich. *From the Second Revised and Enlarged German Edition.* Edited, with additions, by James E. Newcomb, M. D., Instructor in Laryngology, Cornell University Medical School. With 102 illustrations on 42 colored lithographic plates, 41 text-cuts, and 219 pages of text. Cloth, $3.00 net. *In Saunders' Hand-Atlas Series.*

INCLUDING ANATOMY AND PHYSIOLOGY

In designing this atlas the needs of both student and practitioner were kept constantly in mind, and as far as possible typical cases of the various diseases were selected. The illustrations are described in the text in exactly the same way as a practised examiner would demonstrate the objective findings to his class, the book thus serving as a substitute for actual clinical work. The illustrations themselves are numerous and exceedingly well executed, portraying the conditions so strikingly that their study is almost equal to examination of the actual specimens. The editor has incorporated his own valuable experience, and has also included extensive notes on the use of the active principle of the suprarenal bodies in the materia medica of rhinology and laryngology.

Jackson on the Eye

A Manual of the Diagnosis and Treatment of Diseases of the Eye. By EDWARD JACKSON, A. M., M. D., Emeritus Professor of Diseases of the Eye in the Philadelphia Polyclinic. 12mo volume of 535 pages, with 178 beautiful illustrations, mostly from drawings by the author. Cloth, $2.50 net.

In this book more attention is given to the conditions that must be met and dealt with early in ophthalmic practice than to the rarer diseases and more difficult operations that may come later. It is designed to furnish efficient aid in the actual work of dealing with disease, and therefore gives the place of first importance to the conditions present in actual clinical work. A special chapter is devoted to the relations of ocular symptoms and lesions to general diseases.

The Medical Record, New York

"It is truly an admirable work. . . . Written in a clear, concise manner, it bears evidence of the author's comprehensive grasp of the subject. The term 'multum in parvo' is an appropriate one to apply to this work. It will prove of value to all who are interested in this branch of medicine."

Friedrich *and* Curtis' Nose, Larynx, and Ear

Rhinology, Laryngology, and Otology, and Their Significance in General Medicine. By DR. E. P. FRIEDRICH, of Leipzig. Edited by H. HOLBROOK CURTIS, M. D., Consulting Surgeon to the New York Nose and Throat Hospital. Octavo volume of 350 pages. Cloth, $2.50 net.

INCLUDING THEIR SIGNIFICANCE IN GENERAL MEDICINE

In this work the author's object has been to point out the interdependence between disease of the entire organism and diseases of the nose, pharynx, larynx, and ear, and to incorporate the new discoveries of these specialties into the scheme of general medicine. The author has endeavored to bring to the attention of the general practitioner special symptoms and methods of the greatest importance to him.

Boston Medical and Surgical Journal

"This task he has performed admirably, and has given both to the general practitioner and to the specialist a book for collateral reference which is modern, clear, and complete."

Ogden on the Urine

Clinical Examination of Urine and Urinary Diagnosis. A Clinical Guide for the Use of Practitioners and Students of Medicine and Surgery. By J. BERGEN OGDEN, M. D., Late Instructor in Chemistry, Harvard University Medical School; Formerly Assistant in Clinical Pathology, Boston City Hospital. Octavo, 418 pages, 54 illustrations, and a number of colored plates. Cloth, $3.00 net.

SECOND REVISED EDITION — JUST ISSUED

In this edition the work has been brought absolutely down to the present day. Important changes have been made in connection with the determination of Urea, Uric Acid, and Total Nitrogen; and the subjects of Cryoscopy and Beta-Oxybutyric Acid have been given a place. Special attention has been paid to diagnosis by the character of the urine, the diagnosis of diseases of the kidneys and urinary passages; an enumeration of the prominent clinical symptoms of each disease; and the peculiarities of the urine in certain general diseases.

The Lancet, London

"We consider this manual to have been well compiled; and the author's own experience, so clearly stated, renders the volume a useful one both for study and reference."

Vecki's Sexual Impotence

The Pathology and Treatment of Sexual Impotence. By VICTOR G. VECKI, M. D. From the Second Revised and Enlarged German Edition. 12mo volume of 329 pages. Cloth, $2.00 net.

THIRD EDITION, REVISED AND ENLARGED

The subject of impotence has but seldom been treated in this country in the truly scientific spirit that its pre-eminent importance deserves, and this volume will come to many as a revelation of the possibilities of therapeutics in this important field. The reading part of the English-speaking medical profession has passed judgment on this monograph. The whole subject of sexual impotence and its treatment is discussed by the author in an exhaustive and thoroughly scientific manner. In this edition the book has been thoroughly revised, and new matter has been added, especially to the portion dealing with treatment.

Johns Hopkins Hospital Bulletin

"A scientific treatise upon an important and much neglected subject. . . . The treatment of impotence in general and of sexual neurasthenia is discriminating and judicious."

American Pocket Dictionary **Fourth Edition, Revised Just Issued**

The American Pocket Medical Dictionary. Edited by W. A. Newman Dorland, M. D., Assistant Obstetrician to the Hospital of the University of Pennsylvania. Containing the pronunciation and definition of the principal words used in medicine and kindred sciences. Flexible leather, with gold edges, $1.00 net; with thumb index, $1.25 net.

James W. Holland, M. D.,

Professor of Medical Chemistry and Toxicology, and Dean, Jefferson Medical College, Philadelphia,

"I am struck at once with admiration at the compact size and attractive exterior. I can recommend it to our students without reserve."

Stelwagon's Essentials of Skin **Fifth Revised Edition**

Essentials of Diseases of the Skin. By Henry W. Stelwagon, M. D., Ph.D., Clinical Professor of Dermatology in Jefferson Medical College and Women's Medical College, Philadelphia. Post-octavo of 276 pages, with 72 text-illustrations and 8 plates. Cloth, $1.00 net. *In Saunders' Question-Compend Series.*

The Medical News

"In line with our present knowledge of diseases of the skin. . . . Continues to maintain the high standard of excellence for which these question compends have been noted."

Wolff's Medical Chemistry **Fifth Edition, Revised**

Essentials of Medical Chemistry, Organic and Inorganic. Containing also Questions on Medical Physics, Chemical Physiology, Analytical Processes, Urinalysis, and Toxicology. By Lawrence Wolff, M. D., Late Demonstrator of Chemistry, Jefferson Medical College. Revised by Smith Ely Jelliffe, M. D., Ph.D., Professor of Pharmacognosy, College of Pharmacy of the City of New York. Post-octavo of 222 pages. Cloth, $1.00 net. *In Saunders' Question-Compend Series.*

New York Medical Journal

"The author's careful and well-studied selection of the necessary requirements of the student has enabled him to furnish a valuable aid to the student."

Martin's Minor Surgery, Bandaging, and the Venereal Diseases **Second Edition, Revised**

Essentials of Minor Surgery, Bandaging, and Venereal Diseases. By Edward Martin, A. M., M. D., Professor of Clinical Surgery, University of Pennsylvania, etc. Post-octavo, 166 pages, with 78 illustrations. Cloth, $1.00 net. *In Saunders' Question-Compend Series.*

The Medical News

"The best condensation of the subjects of which it treats yet placed before the profession."

Jelliffe and Jackson's Chemistry

A Text-Book of Chemistry. By Smith Ely Jelliffe, M. D., Ph.D., Professor of Pharmacognosy, College of Pharmacy of the City of New York; and Holmes C. Jackson, M. D., Assistant in Chemistry, University and Bellevue Hospital Medical College, N. Y. Octavo, 550 pages, illustrated. *In Preparation.*

Wolf's Examination of Urine

A Laboratory Handbook of Physiologic Chemistry and Urine-examination. By Charles G. L. Wolf, M. D., Instructor in Physiologic Chemistry, Cornell University Medical College, New York. 12mo volume of 204 pages, fully illustrated. Cloth, $1.25 net.

British Medical Journal

"The methods of examining the urine are very fully described, and there are at the end of the book some extensive tables drawn up to assist in urinary diagnosis."

Jackson's Essentials of Eye — **Third Revised Edition**

Essentials of Refraction and of Diseases of the Eye. By Edward Jackson, A. M., M. D., Emeritus Professor of Diseases of the Eye, Philadelphia Polyclinic. Post-octavo of 261 pages, 82 illustrations. Cloth, $1.00 net. *In Saunders' Question-Compend Series.*

Johns Hopkins Hospital Bulletin

"The entire ground is covered, and the points that most need careful elucidation are made clear and easy."

Gleason's Nose and Throat — **Third Edition, Revised**

Essentials of Diseases of the Nose and Throat. By E. B. Gleason, S. B., M. D., Clinical Professor of Otology, Medico-Chirurgical College, Philadelphia, etc. Post-octavo, 241 pages, 112 illustrations. Cloth, $1.00 net. *In Saunders' Question Compends.*

The Lancet, London

"The careful description which is given of the various procedures would be sufficient to enable most people of average intelligence and of slight anatomical knowledge to make a very good attempt at laryngoscopy."

Gleason's Diseases of the Ear — **Third Edition, Revised**

Essentials of Diseases of the Ear. By E. B. Gleason, S. B., M. D., Clinical Professor of Otology, Medico-Chirurgical College, Phila., etc. Post-octavo volume of 214 pages, with 114 illustrations. Cloth, $1.00 net. *In Saunders' Question-Compend Series.*

Bristol Medico-Chirurgical Journal

"We know of no other small work on ear diseases to compare with this, either in freshness of style or completeness of information."

Wolff's Essentials of the Urine

Essentials of Examination of Urine, Chemical and Microscopic, for Clinical Purposes. By Lawrence Wolff, M. D., Late Demonstrator of Chemistry, Jefferson Medical College, Philadelphia. Post-octavo, 66 pages, illustrated. Cloth, 75 cents net. *In Saunders' Question Compends.*

Brockway's Medical Physics — **Second Edition, Revised**

Essentials of Medical Physics. By Fred. J. Brockway, M. D., Late Assistant Demonstrator of Anatomy, College of Physicians and Surgeons, New York. Post-octavo, 330 pages; 155 fine illustrations. Cloth, $1.00 net. *In Saunders' Question Compends.*

Medical Record, New York

"It contains all that one need know on the subject, is well written, and is copiously illustrated."

NAME	DATE DUE
Dr. Crawford	FEB 2 1919 MAR 11 1919
G. H. ...	APR 12 1919